

MULTIPLE ADVERSE EXPERIENCES AND CHILD COGNITIVE DEVELOPMENT

by
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ABSTRACT

Background

Children exposed to multiple adversities in their social environment are more likely to have poorer cognitive outcomes. This dissertation examined the relations between multiple adverse experiences in childhood and cognitive development with a particular emphasis on the type of adversity, timing of exposure, and underlying mechanisms.

Methods

For Aim 1, a guiding conceptual framework describing the relationship between multiple adverse experiences and child cognitive development was developed through a systematic review of the literature. For Aim 2, the relations between multiple adverse experiences and measures of child verbal ability, attention and working memory, including the effects of different domains (or types) of adverse experiences as well as the timing of exposure, were examined using a longitudinal cohort of 2976 children followed from birth to nine years. Using this same cohort, Aim 3 examined whether characteristics of the home environment mediated the effect of different domains of adverse experiences on these cognitive outcomes and whether gender moderated this process.

Results

The conceptual framework proposed from the literature review in Aim 1 presented three domains of adverse experiences, including lack of safety, family instability and economic hardship which were all hypothesized to influence children's

general cognitive ability and executive function by impairing the safety, stability, nurturance and stimulation a child receives in the home environment. In Aim 2, exposure to adversity in infancy and at age three directly predicted cognitive outcomes at ages five and nine, even after controlling for concurrent adverse exposures. Furthermore, economic hardship had the most salient effects. Results from Aim 3 showed that the availability of reading materials and to a lesser extent maternal warmth at age three partially mediated the effect between economic hardship at infancy and cognitive outcomes at ages five and nine. The availability of reading materials also partially mediated the effect between lack of safety and family instability on verbal ability, for boys only.

Conclusions

Efforts to promote safe, stable, nurturing and stimulating home environments through early interventions are promising strategies to improve outcomes for youth. Additionally, policies that alleviate poverty and boost maternal education may positively impact future generations.

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“You are at my side, dear friends, and God is everywhere. Yet ultimately we are alone, making our way home by the candle of the heart. The light is steady and sure but extends only far enough to see the next step. Many times the light seems to go out. But another light, one held by a stranger or friend, a book or a song, a blackbird or a wild flower, comes close enough so that we can see our path by its light. And in time we realize that the light we have borrowed was always our own.”

~Joan Borysenko

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CHAPTER 1

AN INTRODUCTION TO MULTIPLE ADVERSE EXPERIENCES AND CHILD COGNITIVE DEVELOPMENT

DISSERTATION OVERVIEW

The foundations for lifelong health originate in the early years of a child's life (1,2). Healthy people typically grow up in families characterized by nurturing relationships that instill in a child a sense of emotional security, physical safety, and well-being (3). Such families foster the achievement of key developmental milestones that prime the child for lifelong health, including an ability to self-regulate behaviors, emotions and attention, the capacity for communication and learning, and the ability to relate well to others (3-5).

Many families, however, do not fit this description, and children are often raised under more adverse conditions. *Adverse experiences* broadly refer to events or exposures outside of a child's control that are perceived to cause the child excessive harm, threat or uncertainty. These include experiences of abuse, neglect, family instability, parental mental illness, parental substance abuse, parental incarceration, domestic violence, low socioeconomic status, and exposure to neighborhood violence. The majority of research on adverse experiences focuses on the unique contributions of individual adversities on developmental outcomes. However, adverse experiences often co-occur (6-8). Children who face multiple adversities have been shown to fare less well developmentally, and exposure to multiple adversities may explain, in part, the strong relationship between socioeconomic status (SES) and health (4,9-13).

This dissertation builds upon a growing field of research examining the effect of multiple adverse exposures on child health and development, with a specific focus on child cognitive development—a critical aspect of academic success and well-being (14). Although ample evidence suggests multiple adverse experiences are detrimental to child

cognitive development (15-17), there are several gaps in the existing literature that this dissertation aims to address. First, the literature lacks a guiding theoretical framework of the relation between multiple adverse experiences and cognitive development, which is important for informing targets for public health intervention. Second, timing of adverse exposures may matter (2,16,18-20). However, limited studies have examined the influence of timing of multiple adverse exposures on cognitive outcomes (21). Third, the underlying mechanisms through which multiple adverse experiences influence cognitive development are less well understood (21). This dissertation aims to address these gaps.

DISSERTATION ORGANIZATION

This dissertation is organized into six chapters. Chapter 1 describes the dissertation research aims and hypotheses, the background and significance of the topic of multiple adverse experiences and child cognitive development, and guiding theories. Chapter 2 provides detailed information regarding the methods and research design for each of the dissertation aims. Chapters 3-5 contain stand-alone manuscripts for three peer-reviewed publications, each dedicated to addressing one of the three dissertation aims. Finally, Chapter 6 integrates the findings from the three papers and proposes implications and recommendations for future research, policy, and practice.

SPECIFIC AIMS AND HYPOTHESES

The overarching goal of this dissertation was to build upon existing literature, describing the relationship between multiple adverse experiences and child cognitive development, including the influence of timing of adverse exposures on cognitive

development, characteristics of the family that mediate the relationship between adverse exposures and cognitive development, and factors that moderate this process. Three aims and accompanying hypotheses for this dissertation were as follows:

Aim 1: Describe what is known about multiple adverse experiences and child cognitive development through a systematic review of the literature.

Three specific questions were addressed:

- 1) What are the most salient adversities to assess in the context of multiple adverse experiences?
- 2) What is known about underlying mechanisms or mediating pathways between multiple adverse experiences and child cognitive outcomes?
- 3) What is known about the timing of adverse experiences in relation to child cognitive outcomes?

Aim 2: Examine the relationship between multiple adverse experiences and child cognitive outcomes among children in the Fragile Families and Child Wellbeing (FFCW) study.

Drawing upon data from the FFCW study and the results from Aim 1, Aim 2 addressed the following sub-aims and hypotheses:

- Aim 2.1: Evaluate the relationship between cumulative adversity exposure (at infancy and ages three, five and nine years) and cognitive outcomes at ages five and nine years.
 - *Hypothesis 2.1a:* A higher total number of adverse experiences at each age will predict lower cognitive scores (both concurrently and longitudinally).

- *Hypothesis 2.1b*: A higher number of total number of adverse experiences during infancy and at age three will directly predict lower cognitive scores at ages five and nine, even after controlling for adverse experiences at ages five and nine.
- Aim 2.2: Examine whether higher scores in specific adversity domains (i.e., types of adversities) during infancy and ages three, five and nine years differentially effect cognitive outcomes at ages five and nine years.
 - *Hypothesis 2.2a*: Higher adversity domain scores, specifically lack of safety, family instability, and economic hardship, at each age (i.e., infancy, three, five and nine years) will predict lower cognitive outcomes (both concurrently and longitudinally).
 - *Hypothesis 2.2b*: Higher adversity domain scores during infancy and at age three will directly predict lower cognitive scores at ages five and nine, even after controlling for adverse exposures at ages five and nine.
- Aim 2.3: Examine whether exposure to the different adversity domains at age three mediates the relation between exposure to the adversity domains during infancy and cognitive outcomes at ages five and nine. This analysis was purely exploratory. No hypotheses were made.

Aim 3: Identify factors that mediate and moderate the relationship between multiple adverse exposures and child cognitive outcomes among children in the FFCW Study.

Building upon Aims 1 and 2, Aim 3 included the following sub-aims and hypotheses, again, drawing upon data from the FFCW study:

- Aim 3.1: Examine whether characteristics of the home environment, including maternal warmth and availability of reading materials, mediate the relationship between adversity domains (at infancy and age three) and cognitive outcomes (at ages five and nine).
 - *Hypothesis 3.1a*: Availability of reading materials mediates the relationship between the economic hardship domain and cognitive outcomes.
 - *Hypothesis 3.1b*: Maternal warmth mediates the relationship between the adversity domains and cognitive outcomes.
- Aim 3.2: Examine whether gender moderates the mediation process above. This analysis was purely exploratory. No hypotheses were made.

BACKGROUND AND SIGNIFICANCE

Childhood Adversity as a Public Health Problem

Adverse experiences refer to events perceived to cause a child harm, threat or uncertainty. These experiences typically occur outside of the child's control, and may directly or indirectly influence a child's wellbeing (2,22). Although a common operational definition is lacking in the literature, adverse experiences include incidents of childhood maltreatment, trauma or stressful life events (2,22-26). Maltreatment refers most specifically to child abuse (physical, psychological, or sexual) and neglect, whereas trauma also includes witnessing an actual or threatened event that results in intense fear, helplessness, or horror (23,24). Most broadly defined, stressful events include those in which the appraised demands of a situation exceed one's resources, thus evoking anticipation of harm or loss (27). Adverse experiences include, but are not limited to, the

following events: child abuse (physical, sexual or emotional), child neglect, family instability, parental mental illness, parental substance abuse, parental incarceration, low socioeconomic status, and exposure to domestic or community violence.¹

Childhood adversity is common in the United States and associated with a range of health and developmental outcomes throughout the lifespan (1,11). The most common of all childhood adversities is the instability of parental relationships. Family stability refers to whether children are raised with the same parents present from their birth, whether single, cohabitating, or married (28,29). In the United States, approximately 40% of children will experience the divorce of their parents before reaching adulthood (30). An equal percentage of children are born to unwed parents, who are, as a group, more susceptible to instability than married couples (28,29). Other adversities are also common. Twenty-two percent of children grow up in poverty in the United States (31). Before reaching adulthood, 10% of children in the United States will experience some form of child maltreatment, 20% of children will be exposed to domestic violence, and almost a third of children will be exposed to violence in their communities (32). In a given year, 10% of children will experience a mother with depression (33), and 11% of children will live with at least one parent dependent on or abusing alcohol or illicit drugs (34). In 2007, 2.3% of the nation's children had a parent in a state or federal prison (35,36). Extensive research documents the negative effects of each of these individual adversities on a range of psychological, behavioral, and developmental outcomes, including cognitive outcomes and academic success (36-51).

¹ In the literature, studies of multiple adverse experiences often fail to distinguish between adverse exposures, such as those described here, and other risk factors that may be less amenable to public health intervention, including biological risks or genetic predispositions. This dissertation refers to experiences or factors that fall outside of this definition of adversity as "risks" in order to make this distinction.

Multiple Adverse Experiences

The sum of this research clearly demonstrates that individual adverse experiences in early life are associated with a range of health and developmental consequences. However, adversities rarely occur in isolation (7,11,52). Recent data from the National Survey of Children's Health show that nearly one half of children ages 0-17 experienced at least one adversity, and a quarter experienced two or more (1).² Other studies have indicated that 90% of adults who reported exposure to any single adverse experience in childhood also reported exposure to at least one other adversity (7,11). While prevalence estimates range, they indicate that multiple exposures are fairly common. Thus, studies that fail to account for multiple adverse exposures may falsely attribute findings to a single adverse experience when, in fact, one or more correlated adversities or the cumulative effect of multiple adversities may underlie observed effects (7,26,53).

Rutter and Sameroff were some of the earliest to suggest that children who experienced multiple adversities were much more likely to have negative developmental outcomes relative to children with any single adverse exposure (4,10). Sameroff and colleagues used a cumulative index, commonly referred to as "cumulative risk", constructed by dichotomizing different risks and adversities (1 = "exposed" and 0 = "not exposed") and then summing the total exposures into a single aggregated measure.³ They found that verbal IQ in four year olds decreased dramatically as the cumulative risk score

² Adversities measured in the National Survey of Children's Health included: experienced economic hardship, parents divorced or separated, lived with someone with alcohol or drug problem, witnessed or was a victim of neighborhood violence, lived with someone who was mentally ill or suicidal, witnessed domestic violence, parent served jail time, treated or judged unfairly due to race or ethnicity, death of a parent.

³ Researchers use different terminology when referring to multiple adverse experiences, which is reflective of the variability in approaches used across the literature. Given that many researchers use the term "cumulative risk" which may combine both measures of adversity as well as other risk factors, this dissertation refers to the terminology of the original researchers. However, our preference is for the term "cumulative adversity" or "cumulative index," which we use to refer to our own research.

increased (10).⁴ Different combinations of exposures produced similar effects, suggesting that there were no unique effects from any single risk or adversity alone. This same study sought to understand whether these exposures, common to conditions of poverty, explained the relationship between low SES and IQ. A similar effect was observed for both high and low SES groups, though low SES children fared slightly worse than high SES children for a given cumulative risk score. The cumulative index also explained more of the variability in children's IQ than did SES alone.

Another seminal study, the Adverse Childhood Experiences Study (or ACE Study), examined the association of multiple adverse experiences in childhood with numerous leading causes of death among adults in the United States (11). The ACE Study used a cumulative score comprised of ten different adversities.⁵ In a retrospective assessment of nearly 15,000 middle-class, adult Kaiser Permanente patients, the ACE Study showed that adversities before the age of 18 years were common, they frequently co-occurred, and as the number of adversities increased, so too did the likelihood of disease (including ischemic heart disease, cancer, chronic lung disease, and sexually transmitted infections), common risk factors for disease (including smoking, obesity, and substance use), mental health problems (including depression, anxiety, and sleep disturbances), and sexual and reproductive health problems (including early sexual initiation, teen pregnancy, and unintended pregnancy) (11,54-56).

⁴ Risk factors included maternal mental health, maternal anxiety, head of household occupation, maternal education, parenting perspectives, maternal interaction, minority group status, family social support, family size, and stressful life events. Each variable was dichotomized with the upper quartile designated as being at risk.

⁵ The original 1998 ACE Study publication only assessed seven ACE categories, including: three categories of abuse (psychological, physical, and sexual) and four categories of household dysfunction (mother treated violently, household substance abuse, household mental illness, and incarcerated household member). Parental separation or divorce and two categories for neglect (physical and emotional) were added in later ACE studies.

Studies assessing health and developmental outcomes associated with multiple, co-occurring risk factors have since become more prevalent; the majority of these studies have used an aggregate cumulative index to assess a large number of correlated risk factors or adversities. A recent systematic review by Evans and colleagues summarized studies of cumulative risk and child development and found significant main effects of cumulative risk on a number of developmental outcomes, including cognitive development, academic achievement, internalizing and externalizing behaviors, social competencies and self-regulatory behavior (21). Several studies have also suggested that multiple adverse exposures explain, in part, the association of SES with negative health outcomes (9,10,57,58).

Adversity and Child Cognitive Development

Although the development of social, emotional and cognitive abilities are all essential for a child's success and wellbeing, this dissertation focuses more narrowly on the influence of multiple adverse experiences on child cognitive development, and specifically general cognitive ability and executive function. General cognitive ability (often referred to as intellectual capacity or IQ) refers to general reasoning and thinking ability and is associated with one's ability to plan and solve problems (59). Executive functions are one aspect of general cognitive ability, but include distinct neurocognitive processes such as sustained attention, working memory and impulse control (60,61). These processes are integral to the development of behavioral self-regulation and social and cognitive competence (61).

Whereas cognitive development was once thought to be largely the product of genetics, it is now clear that social context and experience play a significant role in shaping cognitive outcomes (59,62). The malleability of cognitive abilities in response to social context is thought to track with brain development. For example, executive functions such as working memory, attention allocation and impulse control are cognitive processes associated with the prefrontal cortex region of the brain (63). Developmental studies have shown these functions develop throughout childhood and adolescence, during which time considerable development and organization of the prefrontal cortex occurs (63). Other aspects of memory, particularly implicit and explicit memory, which include the ability to form new associations among events, are supported by the hippocampus – an area also rapidly developing in early childhood, from birth to age two (14,16).

Given the malleability of the developing brain in response to social context as well as the strong relationship between cognitive development and future success, there is increasing interest in understanding how experience shapes the developing brain and associated cognitive functions. Most studies have looked explicitly at the effects of SES or maltreatment on cognitive function and have shown negative effects. However, a growing number of studies are focusing more comprehensively on the influence of multiple adverse exposures on cognitive development and have shown associations with lower intelligence (64-67), worse academic achievement and school readiness (13,68-71), and poorer attention (13,15,71).

Gaps in the Literature

Atheoretical Framework. While it is generally understood that multiple adverse exposures are detrimental to child cognitive development, there are several limitations to the current state of this research that this dissertation aims to address. Studies of multiple adverse experiences and child cognitive development lack a guiding theoretical framework upon which to inform both research and intervention. As a result, some have labeled this field as atheoretical (21). This is, in part, due to shortcomings in the measurement and analysis of multiple adverse experiences. For example, most studies (like the seminal studies cited earlier) use a cumulative index to examine the influence of multiple adverse experiences on child cognitive outcomes. However, little attention has been paid to the fact that the number and types of adversities vary across studies, and that the adversities are all weighted equally and assumed to be interchangeable in a cumulative metric, regardless of how related or duplicative the different factors may be (21). Some studies focus on an array of adversities across economic, demographic, psychosocial, and neighborhood domains while others are more limited in scope. Additionally, some researchers may classify a certain factor as an adversity whereas others designate the same factor as a covariate or as a mediator between adversities and some developmental outcome. Variables like SES or race, for example, may be considered adversities in some studies and covariates in others; lack of parental warmth or cognitive stimulation are sometimes considered adversities and other times mediating factors (21).

Furthermore, the designation of adversity exposure is often based on sample distribution rather than theory (21). For example, in the 1987 Sameroff study described

above, children were considered exposed to a given factor if they were in the upper quartile of a statistical distribution. Such cut-offs conflate rarity with risk and are not generalizable to other samples. These limitations make it difficult to determine those adversities most salient for developmental outcomes as well as the their underlying mechanisms of action.

Despite these limitations, there are several strengths to the cumulative risk approach. Perhaps simplicity is its greatest strength – one summary score that tallies the number of childhood exposures is an easy metric to understand and communicate to laypersons and policy makers (13,21). Studies using a cumulative index are particularly effective at measuring the effect of dosage of multiple adverse exposures. They are also often more tenable as they preserve statistical power in small samples and avoid issues of collinearity that may present in models that examine multiple, individual adversities that are often highly correlated (13,21).

Evans, Li and Whipple (2013) propose the use of domains as an alternative to a cumulative index (21). Domains are created by aggregating adversities of a similar type or context into a number of groups. For example, an economic hardship domain could be formed by aggregating measures of poverty, food insecurity and housing insecurity. A domain-based approach is promising in that it leverages the advantages of a cumulative measure while also providing additional insight into the salience of particular domains or relations between domains. Consequently, a domain-based approach to the study of multiple adversities also allows for more theoretically driven models linking specific types of adverse exposures to developmental outcomes. Chapter 3 delves into these

issues in more depth, applying the suggestions from the Evan's paper to the study of cognitive development.

Developmentally Sensitive Periods. A second limitation to the current knowledge base is that most studies examining the effects of early adversity on developmental outcomes have disregarded the effect of timing of the exposure (21,72). Research from both animal and human models indicate that during times of rapid development, specific regions of the brain may be more sensitive to environmental threats (16). Stress neurobiology suggests that the primary regions of the brain that respond to conditions of stress are also those that support key cognitive abilities; an overactive stress response system may impair these cognitive functions through cumulative damage over time, or by altering development during critical or sensitive periods (16). Other research suggests that the effects of adversity are strongest in early childhood because of the disruption of parenting behaviors, such as the amount of stimulation in the home, which are critical for healthy development (73).

Most studies of multiple adversities are cross-sectional or fail to assess adversities at multiple points in time, and more longitudinal designs are needed. In studies of behavioral outcomes, there is some evidence that timing of exposure matters, and that adversities in early childhood are more detrimental (13,43,74,75). One study assessed the effect of cumulative indices in early and middle childhood (each made up of five adversities) on adolescent outcomes and found that cumulative risk in early, but not middle, childhood predicted poorer internalizing and externalizing behaviors (74). Another examined the effects of maltreatment timing on education outcomes and found

that first maltreatment prior to kindergarten resulted in worse outcomes compared to first exposure post-kindergarten (76). One study found that a cumulative risk measure in infancy explained more of the variance in school readiness at age five than a cumulative index measure at age three (13). More evidence for early sensitive periods comes from a study showing that the degree of harshness and unpredictability in a child's environment before the age of five years was more likely to predict adolescent risk behaviors relative to the same exposure from ages five to ten years (75). However, others suggest no effect of the timing of adversity on achievement (77,78).

Underlying Mechanisms. A third limitation is that the underlying mechanisms explaining the relationship between multiple adverse exposures and child cognitive development are as yet not well defined. Mechanisms are critical for informing and focusing intervention efforts to both prevent and ameliorate the negative effects of adverse experiences. The lack of clarity in mechanisms is due, in part, to the way many studies in this field have conceptualized and measured multiple adversities, as described above. Tests of mediational effects with different constructs are necessary to discern if there are one or more shared underlying mechanisms explaining the relationship between multiple and/or specific adverse experiences and child cognitive development (21).

Several factors have emerged from the literature to explain the relationship between multiple adverse experiences and cognitive outcomes. Stress neurobiology is one proposed mechanism by which early adversity may influence cognitive development (20), and a growing number of studies have shown that early adversity is associated with heightened stress reactivity measured on the biological level (79,80). Additionally, there

is evidence to suggest that early stress is associated with impaired memory, mediated by structural changes in the developing prefrontal cortex (81,82).

Adversities may also interfere with a parent's ability to provide a nurturing and responsive environment. Maternal responsiveness has emerged as a potential mediator between multiple adversities and cognitive development; specifically, the confluence of multiple adversities has been associated with less maternal responsiveness and warmth (13,21,79). Parental responsiveness is also associated with child attachment such that more responsive parenting yields more securely attached children. Attachment security is also thought to influence stress reactivity; more securely attached children exhibit less reactivity to acute stressors (3,79,81).

Lack of cognitive stimulation is another pathway by which adversity may influence a child's cognitive development. Parental investments during early childhood years through cognitive stimulation in the home may significantly impact child brain development, thus affecting cognitive skills (13,64,83). Therefore, adversities that interfere with or disrupt such parenting practices may influence child cognitive outcomes.

There may also be effects of other biological factors, such as genetics, low birth weight, or prenatal maternal behaviors and exposures, as well as maternal age and education (83-86).

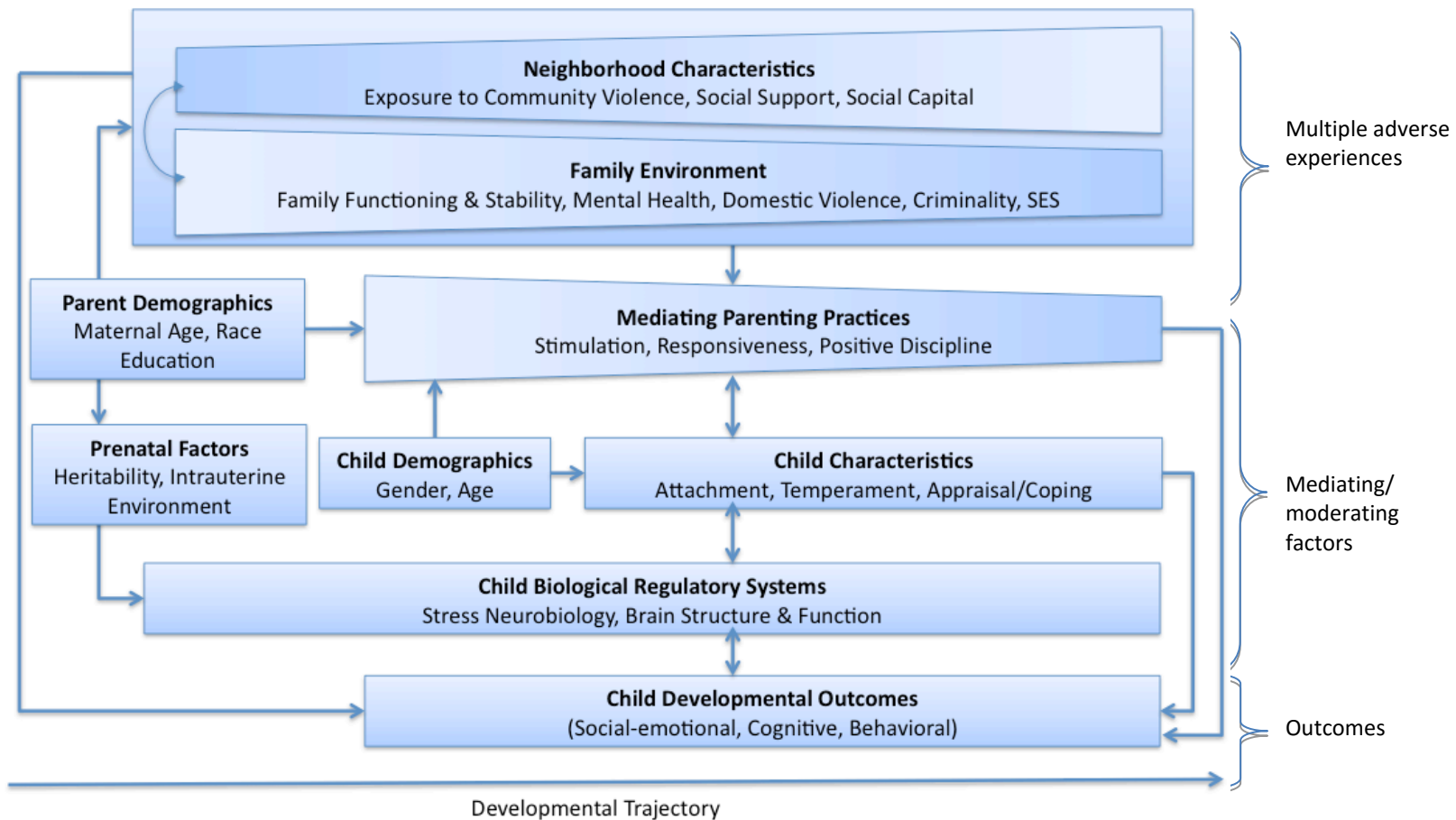
THEORETICAL AND CONCEPTUAL FRAMEWORKS

Conceptual Framework

Figure 1.1 illustrates potential pathways by which multiple adversities influence child developmental outcomes (this framework is developed further in Chapter 3). This

conceptual framework draws from the bioecological theory of human development, stress neurobiology, and attachment theory, all described in more detail below (87-89). Overall, nested levels of a child's environment, particularly the family and neighborhood, influence developmental outcomes through mediating processes, including parenting practices, child characteristics, and biological regulatory systems. Adversities occur in the context of the family and neighborhood. Whereas the family environment has greater influence on child development in early life, characteristics of the neighborhood and social context gain increasing influence on development across the lifespan. A number of other factors may confound or moderate the relationship between adverse exposures and child outcomes. These include prenatal factors and parent and child demographics.

Figure 1.1. Conceptual Model Illustrating Pathways of Influence of Multiple Adversities on Child Development



Bioecological Theory of Human Development

Bronfenbrenner's bioecological theory of human development posits that human development is comprised of the interactions, *or proximal processes*, between an individual and the most immediate, frequently experienced aspects of their environment over time (87). In early childhood, adversity in a child's family environment may negatively impact cognitive development through mediating processes of impaired parenting (e.g., lack of cognitive stimulation and/or responsiveness). As well, such family adversities may also be influenced by the broader neighborhood context (13,84,90). Thus, the confluence of multiple adversities is likely to disrupt proximal processes necessary for healthy child development, overwhelming the child's adaptive capacities, leading to delay and/or dysfunction. In contrast, exposure to any single adversity may be more easily overcome through alternative adaptive processes (21,91).

Stress Neurobiology

Although adversity may disrupt parenting practices that lead to healthy child development, the experience of adversity is also directly stressful, affecting a child's underlying physiology. A review of stress neurobiology and brain structure and function thus serves to explain how early adversities may ultimately influence cognitive function in the developing brain. The concepts of allostasis and allostatic load provide a framework to explain the underlying process (18,19,88,92). Allostasis describes the process by which the human body maximizes survival and maintains stability, or homeostasis, amidst change (18,92,93). In response to threat, sensory information from the environment is translated into a set of cognitive, behavioral, and physiological

responses that are critical to survival; continuous engagement of the stress response, however, may inhibit cognitive, behavioral, and physiological adaptation in the long term (16,81,94).

One of these responses, the hypothalamic-pituitary-adrenal (HPA) stress response is well-studied (16). Cortisol, the end-product of the HPA cascade, acts throughout the body and brain (16,94,95). Cortisol receptors are densely expressed in the hippocampus, prefrontal cortex, and amygdala – regions of the brain that regulate the HPA axis (16). Animal and human models suggest that overproduction of cortisol in response to chronic stress and the underproduction of cortisol that may arise from severe deprivation can inhibit neurogenesis in the hippocampus and the prefrontal cortex, negatively impacting learning, memory, and cognition (16,20,81,94). These brain regions develop rapidly in childhood, and may, therefore, be more vulnerable to the effects of chronic stress or adversity (16,19,96). Since the hippocampus, known to influence memory, develops most rapidly from birth to age two, earlier exposure may have more detrimental effects (16). The prefrontal cortex develops over an extended period of time, and therefore, vulnerability to the effects of chronic stress may occur into and through adolescence (16).

Attachment Theory

Attachment theory, based on the work of John Bowlby and Mary Ainsworth, posits that infants and children have an inherent, biological motivation, organized by the central nervous system, to maintain close proximity to their caregiver (usually the mother) (89,97,98). Such proximity ensures survival by allowing a secure space for exploration and a safe haven for protection in the presence of threat (3,98). During times

of stress, a child's attachment system may be activated as a coping strategy, and he/she will seek protection from the attachment figure to quell the response (3,98). While all children become attached to their caregivers (even children of abuse), not all children are securely attached. Securely attached children are thought to have a mental representation of the attachment figure as responsive to their needs, whereas insecurely attached children lack such a representation (98). Studies have documented a significant relationship between adverse experiences and child attachment, and between child attachment and cognitive outcomes (13,15,21). The sum of this research suggests that adverse experiences threaten attachment security by interfering with the child's perception caregiver's responsiveness. Children with less responsive parents and less secure attachment also exhibit higher HPA reactivity to acute stressors compared to securely attached children (3,81,99). Therefore, maternal-child attachment may be one mechanism by which cumulative risk influences child cognitive development.

CONCLUSION

Although mounting evidence points to detrimental effects of early adversity on cognitive development, significant research gaps remain. A more thorough conceptualization of adversities as well as theoretically determined designations of exposure are required. This dissertation addresses this gaps by first conducting a comprehensive review of empirical studies assessing the relationship between multiple adversities and cognitive development in youth (Chapter 3). The primary purpose for this review is to develop an organizing framework that identifies the adverse experiences theoretically linked with cognitive outcomes as well as potential mediating pathways

between these adversities and child cognitive development. The literature review also discusses what is known about whether particular adverse experiences or broader adversity domains have a more dominant influence on cognitive outcomes. Findings are used to conceptualize the adversities used in the analyses for Aims 2 and 3. The relations between different adversity domains and cognitive outcomes are then examined among a cohort of children from the Fragile Families and Child Wellbeing Study (Chapter 4).

There is also a dearth of evidence on the temporal influence of multiple adversities on cognitive outcomes, an area of research important for informing intervention timing. Using data from Fragile Families and Child Wellbeing Study, this dissertation addresses this gap by examining the influence of multiple adversities experienced when children are very young (at infancy and around three years old), in the pre-school phase (five years) and late middle childhood (nine years) on child cognitive outcomes at ages five and nine (in Chapter 4).

Finally, mediating factors that explain the predictive power of multiple adversities on developmental outcomes are not well understood. Although a subset of studies have explored the mediating pathways of multiple adversities on child cognitive development, research in this area can be improved with a stronger theoretical basis upon which to inform the underlying pathways. The dissertation addresses this gap by first synthesizing known and hypothesized mediating factors through a comprehensive literature review (in Chapter 3) and then examining potential mediators of multiple adversities and cognitive development in the analysis of the Fragile Families and Child Wellbeing Study data (in Chapter 5). The sum of these findings and implications for future research, policy and practice are discussed in the concluding chapter (Chapter 6).

REFERENCES

1. Bethell CD, Newacheck P, Hawes E, Halfon N. Adverse childhood experiences: assessing the impact on health and school engagement and the mitigating role of resilience. *Health Affairs* 2014;33:2106–15.
2. Shonkoff J. Building a new biodevelopmental framework to guide the future of early childhood policy. *Child Development* 2010;81:357–67.
3. Repetti RL, Taylor SE, Seeman TE. Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin* 2002;128:330.
4. Rutter M. Protective factors in children's responses to stress and disadvantage. *Annals of the Academy of Medicine, Singapore* 1979;8:324.
5. National Research Council Institute of Medicine. *From Neurons to Neighborhoods: The Science of Early Child Development*. National Academy Press; 2000.
6. Finkelhor D, Turner H, Ormrod R, Hamby SL. Violence, abuse, and crime exposure in a national sample of children and youth. *Pediatrics* 2009;124:1411–23.
7. Dong M, Anda RF, Felitti VJ, et al. The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect* 2004;28:771–84.
8. Felitti VJ, Anda RF. The Lifelong Effects of Adverse Childhood Experiences. In: *Child Maltreatment*. Saint Louis: STM Learning, Inc; 2014;203–16.
9. Evans GW, Kim P. Multiple risk exposure as a potential explanatory mechanism for the socioeconomic status-health gradient. *Annals of the New York Academy of Sciences* 2010;1186:174–89.
10. Sameroff AJ, Seifer R, Barocas R, Zax M, Greenspan S. Intelligence quotient scores of 4-year-old children: social-environmental risk factors. *Pediatrics* 1987;79:343–50.
11. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine* 1998;14:245–58.
12. Dube SR, Felitti VJ, Dong M, Chapman DP, Giles WH, Anda RF. Childhood Abuse, Neglect, and Household Dysfunction and the Risk of Illicit Drug Use: The Adverse Childhood Experiences Study. *Pediatrics* 2003;111:564–72.
13. Mistry RS, Benner AD, Biesanz JC, Clark SL, Howes C. Family and social risk, and parental investments during the early childhood years as predictors of low-income children's school readiness outcomes. *Early Childhood Research Quarterly* 2010;25:432–49.
14. Noble KG, Tottenham N, Casey BJ. Neuroscience perspectives on disparities in school readiness and cognitive achievement. *The Future of Children* 2005;15:71–89.

15. Pasco Fearon RM, Belsky J. Attachment and Attention: Protection in Relation to Gender and Cumulative Social-Contextual Adversity. *Child Development* 2004;75:1677–93.
16. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci* 2009;10:434–45.
17. Lupien SJ, Fiocco A, Wan N, et al. Stress hormones and human memory function across the lifespan. *Psychoneuroendocrinology* 2005;30:225–42.
18. McEwen BS. Early life influences on life-long patterns of behavior and health. *Ment Retard Dev Disabil Res Rev* 2003;9:149–54.
19. McEwen BS, Gianaros PJ. Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences* 2010;1186:190–222.
20. Pechtel P, Pizzagalli DA. Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology* 2010;214:55–70.
21. Evans GW, Li D, Whipple SS. Cumulative risk and child development. *Psychological Bulletin* 2013;139:1342.
22. Burgermeister D. Childhood adversity: a review of measurement instruments. *Journal of Nursing Measurement* 2007;15:163–76.
23. Leeb RT, Paulozzi LJ, Melanson C, Simon TR, Arias I. *Child Maltreatment Surveillance: Uniform Definitions for Public Health and Recommended Data Elements, Version 1.0*. Atlanta (GA): Centers for Disease Control and Prevention, National Center for Injury Control; 2008.
24. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4 ed. Washington, DC: American Psychiatric Association; 2000.
25. Mulvihill D. The Health Impact of Childhood Trauma: An Interdisciplinary Review, 1997-2003. *Issues Compr Pediatr Nurs* 2005;28:115–36.
26. Jacobs J, Agho K, Stevens G, Raphael B. Do childhood adversities cluster in predictable ways? A systematic review. *Vulnerable Children and Youth Studies* 2012;7:103–15.
27. Lazarus R. *Psychological Stress and the Coping Process*. New York: McGraw-Hill International; 1966.
28. Waldfogel J, Craigie TA, Brooks-Gunn J. Fragile families and child wellbeing. *The Future of Children* 2010;20:87.
29. Cavanagh SE, Huston AC. Family instability and children's early problem behavior. *Social Forces* 2006;85:551–81.
30. Amato PR. The consequences of divorce for adults and children. *Journal of Marriage and Family* 2004;62:1269–87.
31. National Center for Children in Poverty. *Child poverty*. Retrieved 2015 Sep 1.

Available from: <http://www.nccp.org/topics/childpoverty.html>

32. Finkelhor D, Turner HA, Shattuck A, Hamby SL. Violence, Crime, and Abuse Exposure in a National Sample of Children and Youth. *JAMA Pediatr* 2013;167:614.
33. Ertel KA, Rich-Edwards JW, Koenen KC. Maternal Depression in the United States: Nationally Representative Rates and Risks. *Journal of Women's Health* 2011;20:1609–17.
34. Substance Abuse and Mental Health Services Administration. The NSDUH Report: Children Living with Substance-Dependent or Substance-Abusing Parents: 2002 to 2007. Rockville, MD: 2009. Retrieved 2015 Sept 2. Available from: <http://www.samhsa.gov/data/2k9/SAparents/SAparents.pdf>
35. Murray J, Farrington DP, Sekol I. Children's antisocial behavior, mental health, drug use, and educational performance after parental incarceration: A systematic review and meta-analysis. *Psychol Bull* 2012;138:175–210.
36. Johnson EI, Easterling B. Understanding Unique Effects of Parental Incarceration on Children: Challenges, Progress, and Recommendations. *Journal of Marriage and Family* 2012;74:342–56.
37. Jacobson SW, Jacobson JL. Alcohol and drug-related effects on development: A new emphasis on contextual factors. *Infant Ment Health J* 2001;22:416–30.
38. Mills R, Alati R, O'Callaghan M, et al. Child Abuse and Neglect and Cognitive Function at 14 Years of Age: Findings From a Birth Cohort. *Pediatrics* 2011;127:4–10.
39. Watts English T, Fortson BL, Gibler N, Hooper SR, De Bellis MD. The psychobiology of maltreatment in childhood. *Journal of Social Issues* 2006;62:717–36.
40. Holt S, Buckley H, Whelan S. The impact of exposure to domestic violence on children and young people: A review of the literature. *Child Abuse & Neglect* 2008;32:797–810.
41. Evans SE, Davies C, DiLillo D. Exposure to domestic violence: A meta-analysis of child and adolescent outcomes. *Aggression and Violent Behavior* 2008;13:131–40.
42. Margolin G, Gordis EB. Co-occurrence between marital aggression and parents' child abuse potential: The impact of cumulative stress. *Violence and Victims* 2003;18:243–58.
43. Fantuzzo JW, Mohr WK. Prevalence and effects of child exposure to domestic violence. *The Future of Children* 1999;9:21–32.
44. Johnson JL, Leff M. Children of substance abusers: Overview of research findings. *Pediatrics* 1999;103:1085–99.
45. Turney K. Pathways of disadvantage: Explaining the relationship between maternal depression and children's problem behaviors. *Social Science Research*

2012;41:1546–64.

46. Goodman SH, Gotlib IH. Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. *Psychological review* 1999;106:458.
47. Smith M. Parental mental health: disruptions to parenting and outcomes for children. *Child & Family Social Work* 2004;9:3–11.
48. Hammen C, Brennan PA. Severity, chronicity, and timing of maternal depression and risk for adolescent offspring diagnoses in a community sample. *Archives of General Psychiatry* 2003;60:253.
49. Cicchetti D, Lynch M. An ecological-transactional analysis of children and contexts: The longitudinal interplay among child maltreatment, community violence, and children's symptomatology. *Development and Psychopathology* 1998;10:1–23.
50. Foster H, Brooks-Gunn J. Toward a Stress Process Model of Children's Exposure to Physical Family and Community Violence. *Clin Child Fam Psychol Rev* 2009;12:71–94.
51. Fowler PJ, Tompsett CJ, Braciszewski JM, Jacques-Tiura AJ, Baltes BB. Community violence: A meta-analysis on the effect of exposure and mental health outcomes of children and adolescents. *Development and Psychopathology* 2009;21:227.
52. Finkelhor D. Improving the Adverse Childhood Experiences Study Scale. *Improving the Adverse Childhood Experiences Scale. JAMA Pediatr* 2013;167:70.
53. Dunn VJ, Abbott RA, Croudace TJ, et al. Profiles of family-focused adverse experiences through childhood and early adolescence: The ROOTS project a community investigation of adolescent mental health. *BMC Psychiatry* 2011;11:109.
54. Anda RF, Croft JB, Felitti VJ, et al. Adverse childhood experiences and smoking during adolescence and adulthood. *JAMA: The Journal of the American Medical Association* 1999;282:1652–8.
55. Anda RF, Felitti VJ, Bremner JD, et al. The enduring effects of abuse and related adverse experiences in childhood. *European archives of psychiatry and clinical neuroscience* 2006;256:174–86.
56. Anda RF, Butchart A, Felitti VJ, Brown DW. Building a Framework for Global Surveillance of the Public Health Implications of Adverse Childhood Experiences. *American Journal of Preventive Medicine* 2010;39:93–8.
57. Wells NM, Evans GW, Beavis A, Ong AD. Early childhood poverty, cumulative risk exposure, and body mass index trajectories through young adulthood. *Journal Information* 2013;100:1–8.
58. Evans GW, English K. The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development*

2003;73:1238–48.

59. Nisbett RE, Aronson J, Blair C, et al. Intelligence: New findings and theoretical developments. *American Psychologist* 2012;67:130–59.
60. Blair C. How similar are fluid cognition and general intelligence? A developmental neuroscience perspective on fluid cognition as an aspect of human cognitive ability. *Behavioral and Brain Sciences* 2006;29:109–60.
61. Blair C, Zelazo PD, Greenberg MT. The Measurement of Executive Function in Early Childhood. *Developmental Neuropsychology* 2005;28:561–71.
62. Nelson CA, de Hann M, Thomas KM. *Neuroscience of Cognitive Development: The Role of Experience and the Developing Brain*. Hoboken, New Jersey: John Wiley & Sons Inc; 2006.
63. Casey BJ, Giedd JN, Thomas KM. Structural and functional brain development and its relation to cognitive development. *Biological psychology* 2000;54:241–57.
64. Klebanov PK, Brooks-Gunn J. Cumulative, Human Capital, and Psychological Risk in the Context of Early Intervention: Links with IQ at Ages 3, 5, and 8. *Annals of the New York Academy of Sciences* 2006;1094:63–82.
65. Burchinal MR, Roberts JE, Hooper S, Zeisel SA. Cumulative risk and early cognitive development: A comparison of statistical risk models. *Developmental Psychology* 2000;36:793–807.
66. Hall JE, Sammons P, Sylva K, et al. Measuring the combined risk to young children's cognitive development: An alternative to cumulative indices. *British Journal of Developmental Psychology* 2010;28:219–38.
67. Sameroff AJ, Seifer R, Baldwin A, Baldwin C. Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development* 1993;64:80–97.
68. Adelman PK. Social environmental factors and preteen health-related behaviors. *Journal of Adolescent Health* 2005;36:36–47.
69. Burke N, Hellman J, Scott B, Weems C, Carrion V. The impact of adverse childhood experiences on an urban pediatric population. *Child Abuse & Neglect* 2011;35.
70. Rouse HL, Fantuzzo JW. Multiple risks and educational well being: A population-based investigation of threats to early school success. *Early Childhood Research Quarterly* 2009;24:1–14.
71. Alaimo K, Olson CM, Frongillo EA. Food insufficiency and American school-aged children's cognitive, academic, and psychosocial development. *Pediatrics* 2001;108:44–53.
72. Bosch NM, Riese H, Reijneveld SA, et al. Timing matters: Long term effects of adversities from prenatal period up to adolescence on adolescents' cortisol stress response. *The TRAILS study. Psychoneuroendocrinology* 2012;37:1439–47.
73. Brooks-Gunn J, Duncan GJ. The effects of poverty on children. *The Future of*

Children 1997;7:55–71.

74. Appleyard K, Egeland B, Dulmen MHM, Alan Sroufe L. When more is not better: the role of cumulative risk in child behavior outcomes. *Journal of Child Psychology and Psychiatry* 2005;46:235–45.
75. Simpson JA, Griskevicius V, Kuo SI-C, Sung S, Collins WA. Evolution, stress, and sensitive periods: The influence of unpredictability in early versus late childhood on sex and risky behavior. *Developmental Psychology* 2012;48:674–86.
76. Fantuzzo JW, Perlman SM, Dobbins EK. Types and timing of child maltreatment and early school success: A population-based investigation. *Children and Youth Services Review* 2011;33:1404–11.
77. Jaffee SR, Maikovich-Fong AK. Effects of chronic maltreatment and maltreatment timing on children's behavior and cognitive abilities. *Journal of Child Psychology and Psychiatry* 2010;52:184–94.
78. Ackerman BP, Izard CE, Schoff K, Youngstrom EA, Kogos J. Contextual Risk, Caregiver Emotionality, and the Problem Behaviors of Six-and Seven-Year-Old Children from Economically Disadvantaged Families. *Child Development* 1999;70:1415–27.
79. Evans G, Kim P, Ting A, Tesher H, Shannis D. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology* 2007;43:341.
80. Lupien S, King S, Meaney M, McEwen B. Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology* 2001;13:653–76.
81. Gunnar M, Quevedo K. The Neurobiology of Stress and Development. *Annu Rev Psychol* 2007;58:145–73.
82. Hanson JL, Chung MK, Avants BB, et al. Structural Variations in Prefrontal Cortex Mediate the Relationship between Early Childhood Stress and Spatial Working Memory. *Journal of Neuroscience* 2012;32:7917–25.
83. Brooks-Gunn J, Gross RT, Kraemer HC, Spiker D, Shapiro S. Enhancing the cognitive outcomes of low birth weight, premature infants: for whom is the intervention most effective? *Pediatrics* 1992;89:1209–15.
84. Schoon I, Bynner J, Joshi H, Parsons S, Wiggins RD, Sacker A. The influence of context, timing, and duration of risk experiences for the passage from childhood to mid-adulthood. *Child Development* 2002;73:1486–504.
85. Aarnoudse-Moens CSH, Weisglas-Kuperus N, van Goudoever JB, Oosterlaan J. Meta-Analysis of Neurobehavioral Outcomes in Very Preterm and/or Very Low Birth Weight Children. *Pediatrics* 2009;124:717–28.
86. Anderson PJ. Executive Functioning in School-Aged Children Who Were Born Very Preterm or With Extremely Low Birth Weight in the 1990s. *Pediatrics* 2004;114:50–7.

87. Bronfenbrenner U, Morris PA. The ecology of developmental processes. In: Lerner RM, editor. *Theoretical Models of Human Development. Handbook of Child Psychology*. New York: John Wiley & Sons Inc; 1998. p. 993–1028.
88. McEwen BS. Physiology and Neurobiology of Stress and Adaptation: Central Role of the Brain. *Physiological Reviews* 2007;87:873–904.
89. Bowlby J. The Nature of the Child's Ties to His Mother. *International Journal of Psycho-Analysis* 1958;39:350–73.
90. Ackerman BP, Brown ED, Izard CE. The Relations Between Contextual Risk, Earned Income, and the School Adjustment of Children From Economically Disadvantaged Families. *Developmental Psychology* 2004;40:204–16.
91. Flouri E, Kallis C. Adverse Life Events and Psychopathology and Prosocial Behavior in Late Adolescence: Testing the Timing, Specificity, Accumulation, Gradient, and Moderation of Contextual Risk. *J Am Acad Child Adolesc Psychiatry* 2007;46:1651–9.
92. McEwen BS. Introduction: Protective and damaging effects of stress mediators: The good and bad sides of the response to stress. *Metabolism* 2002;51:2–4.
93. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior* 2012;106:29–39.
94. McEwen BS. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology* 2008;583:174–85.
95. Gunnar MR, Barr RG. Stress, early brain development, and behavior. *Infants & Young Children* 1998;11:1–14.
96. Nelson CA, Carver LJ. The effects of stress and trauma on brain and memory: A view from developmental cognitive neuroscience. *Development and Psychopathology* 1999;10:793–809.
97. Bretherton I. The origins of attachment theory: John Bowlby and Mary Ainsworth. *Developmental Psychology* 1992;28:759.
98. Cassidy J. The nature of the child's ties. *Handbook of attachment: Theory, research, and clinical applications* 1999;2:3–22.
99. Evans GW, Kim P. Childhood Poverty and Health: Cumulative Risk Exposure and Stress Dysregulation. *Psychological Science* 2007;18:953–7.

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CHAPTER 2

METHODS AND RESEARCH DESIGN

This chapter describes the methods used for each dissertation aim. The methods description for Aim 1 details how the literature review was conducted, the inclusion and exclusion criteria used to select articles for the review, and a summary of studies that met these criteria. Aims 2 and 3 utilize data from the Fragile Families and Child Wellbeing Study. A description of this study is first provided, including the study design, sample and sampling methods, followed by detailed methods for how these data were used to address Aims 2 and 3. An abbreviated description of the methods for each of the three aims can also be found within the corresponding papers for each aim (Chapters 3-5).

AIM I METHODS

Aim 1 of this dissertation was to *describe what is known about multiple adverse experiences and child cognitive development through a systematic review of the literature.*

This review aimed to answer the following questions:

- 4) What are the most salient adversities to assess in the context of multiple adverse experiences?
- 5) What is known about underlying mechanisms or mediating pathways between multiple adverse experiences and child cognitive outcomes?
- 6) What is known about the timing of adverse experiences in relation to child cognitive outcomes?

Literature Search and Retrieval

The study of childhood adversity spans the fields of education, psychology, sociology, medicine and public health. Therefore, the PubMed, PsycINFO, CINAHL, Web of Science, and Scopus databases which span these fields were searched for

variations of the following search strings: [“cumulative adversity” OR “cumulative risk” OR “adverse experience(s)” OR “adverse events” OR “stressful life events” OR “early life stress” OR “multiple risk” OR “adversity” OR “number of risk factors” OR “adverse childhood experience”] **AND** [“cognitive function” OR “cognition” OR “academic achievement” OR “attention” OR “school readiness” OR “memory” OR “learning” OR “IQ” OR “Intelligence” OR “executive function” OR “inhibitory control” OR “cognitive control” OR “fluid intelligence” OR “fluid cognition”] **AND** [“children” OR “youth(s)” OR “child” OR “adolescent(s)”]. Search terms used for each database were carefully documented. Article titles and abstracts from the search were screened for their relevance to the review topic. Relevant studies identified in review articles or in reference lists were also collected and screened for relevancy.

To be included in the review, articles met the following criteria: 1) the study was published in English, in a peer-reviewed journal; 2) the study was published from January 1990 to December 2013; 3) the study assessed the multivariate relationship between three or more adverse experiences and at least one cognitive outcome in children 18 years of age or younger; and 4) the study was a primary empirical report using quantitative methods. Articles were excluded from the review for the following reasons: 1) the article was an editorial, review, or meta-analysis; 2) the article described adversities in the context of war, natural disasters, or developing countries; and 3) non-human studies.

In order to focus more specifically on the relationship between multiple adversities and cognitive development, studies were excluded if they did not use at least one standardized assessment of general cognitive ability (such as intellectual functioning or IQ) or of a specific executive function (i.e., attention, impulsivity, inhibitory control,

executive control, and working memory) with demonstrated validity and reliability.

Studies that looked more broadly at achievement, school readiness, academic outcomes (such as grades or highest grade level achieved) or specific learning problems were also excluded as these are multifactorial and more distal to specific cognitive functions. All studies that met these criteria were reviewed and scored for methodological rigor according to the criteria in Table 2.1 (adapted from the Quality Assessment Tool for Quantitative Studies).⁶ This tool assigns studies a score of 7 to 18; studies that did not meet a score of 10, suggesting they were not sufficiently rigorous, were also excluded from the review.

Across studies that met the final criteria for this review, clear distinctions were not apparent with respect to definitions of adverse experiences, stressful life events and risk factors. Many studies referred broadly to all exposures as “risks”, thus encompassing a range of adverse experiences or events (such as abuse or neglect) as well as specific biological risks (such as gender or genetic vulnerabilities). Given the overarching goal of this review to inform future public health interventions, adversities were distinguished from other risks or confounding factors. Adversities were defined as *exposures that typically create excessive demands or threats to the child but are preventable or amenable to change*, thus lending them to intervention.

Search Results

The combined search results yielded 3999 articles (not excluding duplicates). After screening the titles and abstracts for relevancy, 413 articles were reviewed in

⁶ Developed by the Effective Public Health Practice Project; available at: http://www.ehphp.ca/PDF/Quality%20Assessment%20Tool_2010_2.pdf

further detail to determine if the inclusion and exclusion criteria were met. Of these, 23 articles met the final criteria for inclusion in the review. Table 2.2 summarizes the types of studies included in the review. One article utilized a case-control study design, six were cross-sectional, and sixteen utilized a longitudinal design. With respect to outcomes, 17 studies focused on general cognitive ability and 10 studies focused specifically on executive functions (one study focused on both sets of cognitive outcomes).

To effectively summarize the articles, each article was categorized by: the primary cognitive outcome (general cognitive ability or executive function), the analytical method used to assess the relationship between multiple adversities and the cognitive outcome(s), whether or not the study investigated mediating and moderating factors, and whether or not the study investigated timing of exposures in relation to cognitive outcomes. Articles were then summarized by these categorical groupings.

AIMS 2 AND 3 – FRAGILE FAMILIES AND CHILD WELLBEING STUDY

Aims 2 and 3 drew upon publicly available data from the Fragile Families and Child Wellbeing (FFCW) Study to examine the relationship between multiple adverse experiences and child cognitive development. Aim 2 examined the relationship between the timing of multiple adverse experiences in relation to cognitive outcomes, and Aim 3 examined factors that mediated and moderated the relationship between multiple adverse experiences and cognitive outcomes.

Fragile Families and Child Wellbeing Study Overview

The FFCW Study follows a birth cohort of 4700 children born between 1998 and 2000 from twenty large U.S. cities. The cities were selected from a stratified random sample that took into consideration geographic location, policy environments and labor market conditions. The sampling of individuals occurred in three stages: first cities, then hospitals within cities, then births within hospitals. Children born to unmarried parents were oversampled in order to be representative of non-marital births in each city. The study sample is representative of families with children born to unmarried parents in U.S. cities with a population over 200,000. (1).

Parents were excluded from the FFCW Study if they planned to place the child up for adoption, if the father of the baby was not living, if the parents could not complete the interview in English or Spanish, if the mother was too ill to complete the interview, or if the baby died before the interview could take place. Although the study allowed adolescent mothers to participate, mothers under the age of 18 years were excluded in many hospitals based on hospital policies (1).

Data Collection Procedures

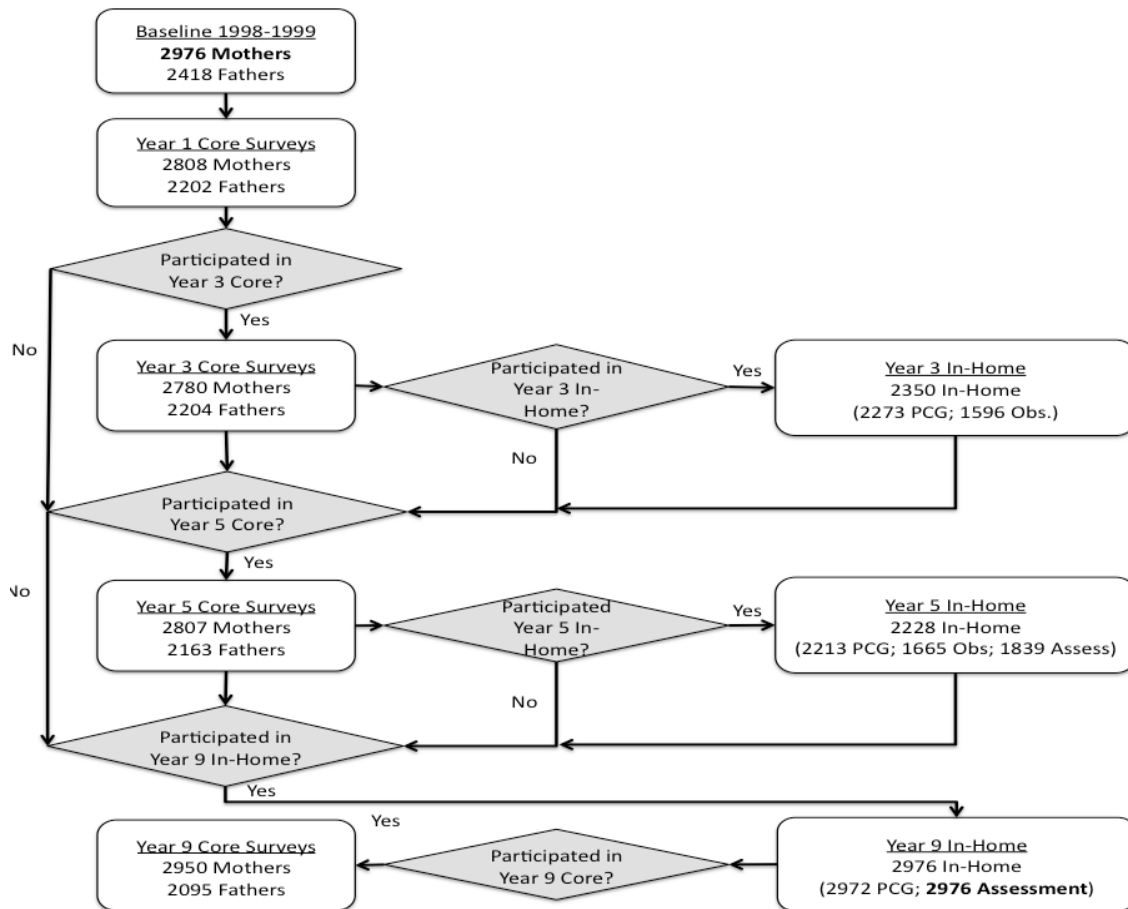
Data collection procedures are illustrated in Figure 2.1. Core Mother and Core Father Surveys were completed with the biological parents by Computer Assisted Telephone Interviewing (CATI) soon after a child's birth (baseline) and when the focal child was one, three, five and nine years of age. At baseline, interviewers attempted to reach both parents as soon after the baby's birth as possible. Mothers were interviewed between 0 and 112 days after their baby's birth, with 99% occurring within the first week

after birth. Fathers were interviewed between 0 and 381 days after their baby's birth, with 77% occurring within the first week after birth. The Core Mother and Core Father Surveys collected information separately from both the biological mother and the biological father on parent attitudes, relationships, parenting behavior, demographics, mental and physical health, economic and employment status, and neighborhood characteristics. While biological father data were not used to generate the final constructed measures, they were used to recover missing data (described in the Missing Data Analysis section).

An In-Home Assessment was conducted when the focal child was three, five and nine years old as part of a FFCW sub-study. The In-Home Assessment was comprised of three components: a Primary Caregiver (PCG) Survey, a Home Observation and a Child Assessment. Only families who participated in the core interviews for each year were eligible for the In-Home Assessment for that same year. However, if a family missed a prior study year for a reason other than refusal to participate, they were still eligible to participate in the subsequent year. The PCG Survey was typically the child's mother, and the survey consisted of an hour-long self-administered questionnaire covering a broad range of topics, including: child's health status, family routines, nutrition, housing characteristics, parenting, child discipline, informal social control and social cohesion and trust, exposure to violence, and child's behavior. In some cases, this survey was conducted over the phone if a primary caregiver refused to have a home visit. When conducted in the home, the PCG Survey was accompanied by the Home Observation and Child Assessment. During the Home Observation, interviewers recorded observations about the home environment, child's appearance, and the parent-child interaction. The

Child Assessment consisted of several cognitive assessments along with measurements of the child's height and weight.

Figure 2.1. Data Collection Procedures and Final Analytic Sample



AIMS 2 AND 3 – DESCRIPTION OF STUDY MEASURES

Aims 2 and 3 utilized data from the Core Surveys (at baseline and when the focal child was one, three, five and nine years old) and the In-Home Assessments (including the PCG survey, Home Observation, and Child Assessment when the focal child was three, five and nine years). A description of the final measures used from each wave of the study is below, and a summary can be found in Table 2.3.

Cognitive Outcomes⁷

Wechsler Intelligence Scale for Children (WISC-IV), Digit Span subtest. The WISC-IV is an intelligence test for children ages 6-16 years designed to measure child cognitive function. The Digit Span subtest of the WISC-IV specifically measures the child's auditory short-term memory, sequencing skills, attention, and concentration. It was obtained only at age nine. Children heard a sequence of numbers and were asked to repeat the numbers either forward or backwards. Scores were aged-normed (standard score of $M=10$, $SD=3$). The subtest has high internal consistency ($\alpha = 0.92$) and high test-retest reliability ($r = 0.89$) (2).

Child Peabody Picture Vocabulary Test (PPVT)-III. The PPVT-III measures receptive vocabulary and screens for verbal ability. At ages five and nine, an interviewer read a word and asked the child to identify the corresponding picture (among a set of four pictures) on an easel. Scores were age-normed (standard score of $M = 100$, $SD = 15$). The PPVT-III has high internal reliability ($\alpha = 0.93$) and test-retest reliability ($r=0.95$) (3).

Sustained Attention and Lack of Impulsivity. The Leiter International Performance Scale—Revised measures children's ability to maintain attention to a specific stimulus over time. At age five, children were shown a picture booklet with a variety of objects placed throughout the page. There was a target object at the top of the page, and children were asked to put a line through as many of the matching target pictures as possible within the allotted time, without erroneously crossing out non-target

⁷ Three measures of achievement, including the Woodcock-Johnson Letter-Word assessment at Y3 and the Woodcock Johnson Passage Comprehension and Applied Problems at Y5 were also explored as cognitive outcomes. However, these outcomes were not used in the final analysis (discussed in more detail under the *Selecting and Constructing Cognitive Outcome Measures* section below).

objects. Average performance across four trials yielded two attention scores. The number of correct responses reflected the child's *sustained attention* whereas the number of incorrect responses (reverse coded) reflected *lack of impulsivity*. Scores were age-normed (standard score of $M=10$, $SD = 3$). The task has high internal reliability ($\alpha = 0.83$) and test–retest reliability ($r = 0.85$) for children 4–5 years of age (4).

Adverse Experiences

Adversity exposures used in this study were selected based on the findings from the Aim 1 literature review as well as available measures in the FFCW Study. Biological mothers reported on the following measures during the core and the primary caregiver interviews at baseline and ages one, three, five and nine. In the first year of life, some of the adversity measures were collected at either baseline or at age one, and therefore, these waves were combined and collectively referred to as *infancy*. Where possible, the same adversity measures were used at each wave of data collection. However, there were a few instances where measures differed across waves, or where adversities were not measured at all waves. These are described in more detail below and summarized in Table 2.4. All of the adversities described below were dichotomized such that 1 = exposed, and 0 = unexposed based on theoretical cut-points. The majority of these measures were used to assess adverse experiences in another FFCW study (5).⁸

Severe Psychological Aggression. The Parent-Child Conflict Tactics Scales (PCCTS) measures child maltreatment and nonviolent modes of discipline by parents (6).

⁸ Maternal drug and alcohol use were also explored as adverse exposures in this study. However, too few respondents in the sample (<1%) reported these exposures, and therefore, these measures were not included in the final analysis.

The 5-item psychological aggression subscale of the PCCTS measures verbal and symbolic acts by the parent intended to cause a child psychological pain or fear. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often they had done the following to the child in the past year: shouted, yelled or screamed at; threatened to spank or hit but didn't actually do it; swore or cursed at; called him or her dumb, lazy or some other name like that; said they would send them away or kick them out of the house. Ordinal responses included "never," "once," "twice," three to five times," etc. Among national samples, approximately 90% of parents report one or more forms of psychological aggression (also reflected in the current study population) (7). However, more severe forms of aggression (swore or cursed at, called dumb or lazy, or threatened to kick out of the house) are less common. Children of mothers who reported that at least one of these *more severe acts* occurred at least once in the last year were categorized as exposed to psychological abuse (prevalence score cut-offs for all PCCTS measures described in Straus et. al., 1998).

Severe Corporal Punishment. Corporal punishment was assessed with a 5-item subscale of the PCCTS. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often in the past year they: spanked the child on the bottom with a bare hand; hit the child on the bottom with something like a belt, hairbrush, a stick or some other hard object; slapped the child on the hand, arm or leg; pinched the child; and shook the child. Ordinal responses included "never," "once," "twice," three to five times," etc. Spanking the child and slapping the child on the arm or leg are considered to be more widely accepted forms of corporal punishment, whereas the other three acts are considered to carry higher risks and be less widely accepted, thus indicating

more severe corporal punishment (8). Children of mothers who reported that at least one of these three *more severe acts* occurred at least once in the last year were categorized as exposed to severe corporal punishment.

Child Neglect. Child neglect was assessed with a 5-item subscale of the PCCTS. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often they: had to leave the child home alone, even when they thought an adult should be with the child; were so caught up with her own problems that they were not able to show or tell the child they loved him/her; were not able to make sure that the child got the food he/she needed; were not able to make sure the child got to a doctor or hospital when needed; had drinking or drugs interfere with taking care of the child. Ordinal responses included “never,” “once,” “twice,” three to five times,” etc. Children of mothers who reported that at least one of these acts occurred at least once in the last year were categorized as exposed to child neglect.

Intimate Partner Violence (IPV). During the core surveys when the child was one, three, five and nine, biological mothers were asked to think about their relationship with the child’s father, or their current partner. For each existing romantic relationship (either with the biological father or a current partner), they were asked previously validated questions (9,10): 1) How often does he slap or kick you?; 2) How often does he hit you with a fist or object that could hurt you?; and 3) How often does he try to make you have sex or do sexual things that you don’t want to? For any relationship with the father (romantic or not) as well as for existing relationships with another current partner, mothers were also asked, “Have you and the father (or current partner) been in a physical fight in front of the child in the time since the last interview?” If mothers answered

“sometimes” or “often” to any of the first three questions, or, “yes” to the last question, they were categorized as experiencing IPV for that time period.

Exposure to Community Violence. Different measures were used to assess exposure to community violence at infancy and the later waves. At infancy, biological mothers were asked during the baseline core survey how safe the streets around their house were at night (very safe, safe, unsafe or very unsafe). Responses of unsafe or very unsafe were categorized as exposure. During the primary caregiver surveys at ages three, five and nine, biological mothers were asked about their own exposure to violence in their neighborhood in the past year. Three questions assessed whether the primary caregivers saw someone get hit, punched, slapped or beaten up by someone else; if they saw someone get attacked with a weapon like a knife or a bat; and if they saw someone get shot. Ordinal responses ranged from never to more than ten times. Exposure to community violence at these waves was defined as at least one exposure to any of these three items.

Parental Relationship Instability. Relationship instability was defined as a change in parental relationship status since the child’s birth (11,12). Prior studies using data from the FFCW Study have shown that children with stable family structures (whether married, cohabitating, or single parents) had better outcomes than children with unstable family structures (characterized by a parent’s partial presence) (13). During the core surveys at baseline and when the child was three, five and nine, biological mothers were asked about their relationship with the biological father. Responses were categorized into: married, cohabitating or single. During the infancy wave, adversity was simply classified as having a single parent family structure at the time of the child’s birth,

as opposed to a married or cohabitating family structure. For the remaining waves, stability was defined as having the same parent structure since the previous wave or moving from a cohabitating relationship to a married relationship since the previous wave. Moving from a married relationship to a cohabitating or single status, or moving from cohabitating relationship to single status was categorized as unstable.

Maternal Depression. The Composite International Diagnostic Interview (CIDI) is a standardized instrument for assessing mental disorders based on DSM-IV criteria. The short form of the CIDI interview takes a portion of the full set of CIDI questions and generates from the responses the probability that the respondent would be a case, if given the full interview (14). When the child was one, three, five and nine, biological mothers were asked all of the essential CIDI-SF questions necessary to classify a major depressive episode. Mothers who met set criteria were classified as probable cases for maternal depression.

Father Incarceration. Father incarceration was determined from both the mother report on the core surveys when the child was one, three, five and nine, and from information collected by interviewers in the field. Mothers were asked whether the father was currently in jail. Fathers were categorized as currently in jail if mothers or interviewers indicated this to be the case.

Living in Poverty. The income to needs ratio adjusts family income by the number of adults and children in the household, using the official poverty thresholds. Absolute poverty is measured by having a poverty ratio less than one. Family income and family size were collected from the biological mother during the core survey at baseline

and ages three, five and nine. Living in poverty was categorized as living below the federal poverty level.

Housing Insecurity. During the core surveys at ages one, three, five and nine, biological mothers were asked four questions derived from the Survey of Income and Program Participation and the New York City Social Indicators Survey (15), including whether they: had been evicted from their home in the past twelve months; stayed in a shelter/car or abandoned vehicle; did not pay full rent or mortgage; or if they had moved in with other people because of financial problems. Mothers responding “yes” to at least one of these questions were categorized as experiencing housing insecurity.

Food Insecurity. During the core survey at age one, the primary caregiver survey at age three, and the core surveys at ages five and nine, biological mothers were asked about whether, in the past twelve months, they were ever hungry but could not afford to buy more food (15). Mothers who responded “yes” to this question were characterized as experiencing food insecurity.

Potential Mediating Factors

Maternal Warmth. Maternal warmth was determined from five yes/no items of the observational Home Observation for Measurement of the Environment (HOME) scales (16) and the Homelife Interview (17) collected during the home visits at ages three and five. Items included: parent talks with child twice during the visit, parent answers child’s questions orally, parent praises child twice during the visit, parent voices positive feelings to child, and parent caresses, kisses, or hugs child. A maternal warmth score was created by summing the five items from each wave ($\alpha = 0.71$ for Y3, $\alpha = 0.63$ for Y5).

Availability of Reading Materials. Availability of reading materials was determined from parental report of two items drawn from the HOME (16) and the Homelife Interview (17) during the age three and five home visits. At age three, mothers reported on the number of adult books in the house (none, 1-9, 10-20, or more than 20) and the number of books for the child in the house (none, 1-2, 3-4, or more than 4). At age five, mothers reported on the number of books in the house (none, 1-9, 10-20, or more than 20) and the number of books and games to help the child learn the alphabet (none, 1-2, 3-4, or more than 4). An availability of reading materials score was created for each year by summing the two items.

Control Variables

Covariates included demographics and other factors associated with child cognitive outcomes.

Child Sex. Child sex was reported on the baseline core mother survey (male/female).

Maternal Race. Mother's self-reported race/ethnicity was used as a proxy for the race/ethnicity of the child (non-Hispanic white, non-Hispanic black, Hispanic, and other).

Maternal Age at Birth. Maternal age at birth (in years) was reported on the mother Core Survey at baseline.

Low Birth Weight. Babies born at a low (≤ 2500 g) or very low (≤ 1500 g) birth weight are at a greater risk for decrements in executive function, attention, and academic achievement (18-20). The Fragile Families public use data set includes a binary variable only denoting low birth weight babies from those who were not based on the mother core

baseline survey. Very low birth weight babies are not identified in the publically available data set.

Twins. A binary variable was constructed to indicate whether the focal child was part of a multiple birth or not. For babies who were part of a multiple birth, only one focal child was included in the study.

Maternal Education at Birth. Maternal education is one of the strongest predictors of child cognitive outcomes (20). Mothers were asked during the baseline Core Survey about their highest level of schooling completed. Categories consisted of less than high school, high school graduate or equivalent, some college or technical degree, and college or graduate school.

Prenatal alcohol use. Mothers were asked during the baseline core survey, how often they drank alcoholic beverages during pregnancy. Responses included “never,” “less than once a month,” “several times a month,” “several times a week,” and “nearly every day.” A binary variable was created to indicate mothers who reported drinking alcohol several times a month or more versus less than once a month.

Prenatal drug use. Mothers were asked during the baseline core survey, how often they used drugs (i.e. marijuana, crack, heroine, or cocaine) during pregnancy. Responses included “never,” “less than once/month,” “several times a month,” “several times a week,” and “nearly every day.” A binary variable was created to indicate mothers who reported any drug use during pregnancy versus none.

Prenatal smoking. Mothers were asked during the baseline core survey, how often they smoked cigarettes during pregnancy. Responses included “none,” “less than 1

pack/day,” “1-2 packs per day,” and “more than 2 packs per day.” A binary variable was created to indicate mothers who reported any smoking during pregnancy versus none.

Maternal Cognitive Ability. Cognitive ability is a function of both genetics and the environment. While the heritability of cognitive abilities has been established, the genetic influence of specific cognitive abilities remains less understood (21,22).

Cognitive ability of biological mothers was assessed during the age three Core Survey using the Similarities subtest from the Wechsler Adult Intelligence Scale – Revised (WAIS-R) (23). The Similarities subtest consists of eight items that measure verbal concept formation and reasoning abilities, though the items may also reflect long-term memory and cultural opportunities. Raw scores ranged from 0-16. The mother’s WAIS-R scores moderately correlated with mother’s education (0.36), and mother’s Peabody Picture Vocabulary Test (PPVT)(0.41) and modestly correlated with the child’s PPVT (0.20).

AIMS 2 AND 3 – ANALYTIC SAMPLE

Sample Selection

From the original sample (N=4789), the analytic sample excluded 132 (3%) families with children who had conditions likely to influence cognitive outcomes: total or partial blindness, total or partial deafness, Down’s syndrome, cerebral palsy, mental retardation or other developmental delay, and autism. Additionally, 1391 (29%) families were excluded because the child did not have at least one cognitive outcome measurement at age nine. To minimize measurement bias, the sample was also limited to

those in which the biological mother completed the PCG survey (as opposed to father or other guardian; 290 excluded (6%)). The final analytic sample included 2976 cases.

Comparison to Full FFCW Sample

Chi square and t-tests (with a significance level of $p < 0.05$) using Stata 13 were used to test the differences between those who remained in the final analytic sample and those who did not (shown in Tables 2.5-2.7). Mothers in the final analytic sample were less likely to be living in poverty and more likely to be educated compared to those excluded from the final analysis, more likely to be non-Hispanic black (50% vs. 43%) and less likely to be non-Hispanic white (20% vs. 23%), Hispanic (27% vs. 29%), or other race (3.4% vs. 5.3%). Mothers who remained in the sample tended to report less exposure to adversity in cases where there were significant differences between those who participated and those who were excluded, which was expected given that the final sample was relatively more advantaged. However, in some cases, participating mothers reported *more* adverse exposure (i.e., severe corporal punishment at age three, father incarceration at age nine, and community violence at age nine). These differences may reflect that these particular adverse exposures tend to be more common among non-Hispanic black populations, which was over-represented in the final analytic sample. Given the significant differences between the full and final analytic samples, the final sample was no longer considered nationally representative of children born to unmarried parents. Although the sample is racially and economically diverse within the low to middle income strata, and families reported a relatively high rate of adverse experiences,

it may not describe the experience of even less advantaged children and non-Hispanic white, Hispanic, and other race families.

AIMS 2 AND 3 – MISSING DATA ANALYSIS

Description of Participant Attrition and Item Non-Response

Sample attrition is described in Table 2.8 and illustrated in Figure 2.1. Over 90% of mothers participated in each wave of the core study. At age three, 76% of mothers also participated in the PCG survey, and 54% of families had a Home Observation. At age five, 74% participated in the primary caregiver survey, 56% of families had a Home Observation, and 62% of children completed the age five Cognitive Assessments. Since inclusion in this sample required data for at least one cognitive outcome at age nine, all mothers participated in the age nine PCG survey and all children participated in the age nine Cognitive Assessment. Eighty-eight percent of the sample participated in all four of the core surveys, 62% participated in all four core surveys and all three waves of the PCG survey, and 37% participated in all four waves of the core survey, all of the PCG surveys, and all of the in-home assessments.

Missingness was also explored for individual variables. As shown in Tables 2.5-2.7, missing data ranged from 0-3% for each of the control variables (except for mother cognitive ability, which was missing 7%), 0% - 26% for the adversity variables, 34% - 44% for the age five cognitive outcomes, ~1% for the age nine cognitive outcomes, and 23-26% for the mediating variables. Overall, the amount of item-level missing data above that due to attrition ranged from 0-5%.

Patterns of Missing Data

In order to determine systematic reasons for missing data, the relationships between observed variables and missing values were examined empirically (24). Since the majority of missing values were due to attrition from a particular wave of data collection rather than item non-response, relationships were first examined between dummy variables representing missingness from a particular wave of the study and the control variables. Relationships were then examined between dummy variables representing missingness on individual items and the control variables to describe missingness beyond that due to attrition. Chi-square tests for categorical variables and t-tests for continuous variables (significance of $p < 0.05$) were used to test the differences between missing and non-missing groups.

Overall, patterns of missingness for each wave of data collection showed that those who participated in the core and PCG surveys were more likely to be educated and less likely to be Hispanic, consistent with earlier trends describing the analytic sample. However, for the in-home assessments, which required a home visit by the interviewer, the opposite was true. Those who participated in the in-home assessments were *less* advantaged (i.e., living below poverty, single, and lower maternal age), which may reflect that more advantaged participants were less likely to agree to an investigator coming to the home.

Specifically, those who participated in the core surveys during infancy and ages three and five were more likely to be non-Hispanic white or black compared to those who were missing. At ages three and five, those who participated were also more likely to have some college education. Those who participated in the PCG surveys at ages three

and five (which took place either by phone or in-home visit) were also more likely to be non-Hispanic white or black. For the age three PCG survey, participants were also more likely to have higher education, and for the age five PCG survey, participants were more likely to have a mother who smoked during the pregnancy, a child born with low birth weight, and higher maternal cognitive scores than those who did not participate. Among those who completed the age three and five Home Observation and the age five Cognitive Assessment (all of which required an home visit), participants were more likely to be non-Hispanic black and less likely to be Hispanic compared to those who did not participate. At age three, participants who completed the Home Observation were also more likely to be living in poverty and to have lower maternal age compared to those who did not do the observation. At age five, those who completed the Home Observation were also more likely to be single and less likely to be married or cohabitating. Only 26 cases were missing from nine-year wave, and these were all from the Core Survey. Those who participated in the age nine core survey were more likely to be white and less likely to be identified as other race.

With respect to missingness of individual variables, the majority of missing data was due to participant attrition from a particular wave. Therefore, patterns of missing data for individual variables reflect the patterns described above. Where there were additional relationships between missingness and observed variables beyond what was expected due to attrition, these relationships showed that those who responded were more likely to be married, living above the poverty level, and with higher maternal cognitive scores. Again, these relationships are consistent with those noted above, suggesting that the final sample was more advantaged than those who did not participate. There were

some adversities (particularly the age three PCCTS measures and age three father incarceration) for which those who were missing were *more* likely to be non-Hispanic black, which is inconsistent with the prior observed patterns of missing data and suggests that non-Hispanic blacks were less likely to respond to these items despite participation in the wave. In addition, although there were fewer missing data for the age nine measures (1-7%), those with data on these measures were more likely to be white or Hispanic and less likely to be black, compared to those who were missing.

Missingness on individual variables was also examined in relation to the age nine cognitive outcomes to determine how missingness might bias the outcomes under study. While there were no significant differences in the age nine Digit Span score among the missing and non-missing groups for all variables, there were several significant relationships associated with the PPVT-III. In all cases where there were significant relationships, missingness on a particular variable was associated with *lower* cognitive scores. This is to be expected among this analytical sample given the missingness was associated with less advantaged populations.

Missing Data Mechanism

One must determine the mechanisms of missing data in order to select the best approach for handling the missing data appropriately. Missing completely at random (MCAR) suggests that the data are truly missing truly at random. Missing at random (MAR) indicates that there is a known reason why data are missing and that observed variables in the data set can predict missingness. Not missing at random (NMAR)

represents data that are missing for an unknown reason or that a certain level of the missing variable is associated with missingness (25).

The data for this study were missing at random (MAR) because factors associated with missingness were observed in the data set (such as race/ethnicity, education, poverty and marital status). Although the MAR mechanism introduces bias, this bias is recoverable with modern missing data methods (described in more detail below) (25). There is the possibility that the MNAR mechanism may be present in these data as well. For example, participants exposed to a particular adversity, such as domestic violence or severe corporal punishment, may be less likely to answer a question about that exposure. However, these instances were more rare, as item non-response on these more sensitive measures represented less than 5% of missing data.

Data Recovery Methods

Modern data recovery methods, such as multiple imputation (MI) or full-information maximum likelihood (FIML), are recommended for handling greater than 5% missing data (25). FIML was used for handling missing data in these analyses. FIML is a model-based approach that uses all of the available data to estimate the parameters of the statistical model in the presence of missing data. FIML is different from MI, which is a data-based approach where missing values are imputed multiple times, pooled together, and then used to generate unbiased estimates of a statistical model. Simulation studies of MAR data verify that when correlates of the reasons for missing data are observed and included in analysis or imputation models, both FIML and MI produce unbiased estimates of model parameters and standard errors. Even for MNAR

data, these missing data methods result in less biased estimates if the data contain observed measures that are known to approximate the unknown reasons for missing data (25).

The degree to which missing data can be recovered with FIML depends upon the extent to which observed variables are both associated with the missingness of a given variable as well as the missing variable itself (25). As described in the section above, overall missingness in this analytic sample was associated with a number of observed variables in the data set, including maternal education, race/ethnicity, maternal cognitive scores, poverty, and marital status at birth. Variables with missing data were also correlated with other observed variables – including observed variables in the analytical model as well as other auxiliary variables. Auxiliary variables are peripheral to the substantive analysis, thus not included in the actual statistical model, but provide information about the reasons for missingness. Whether observed correlates of variables with missing data are used in the analysis model or as auxiliary variables, they will facilitate data recovery by reducing bias (26).

Auxiliary variables with strong face validity were selected from both the mother and father data. Appropriate correlations (Pearson's correlations for continuous variables, polychoric correlations for polytomized variables, and tetrachoric correlations for dichotomized variables) were evaluated; a cut-off of correlation greater than or equal to 0.2 was used to select auxiliary variables. Nine auxiliary variables were identified with correlations ranging from 0.20 – 0.55. These included mother's cognitive ability, father's education level and cognitive ability, whether the mother lived in public housing, relationship status at each wave, and whether the mother was afraid to let the child

outside. In addition to these auxiliary variables, variables with missing data were also correlated with other variables in the model (correlations ranging from 0.21 – 0.80). In cases where the data were suspected to be partly MNAR (i.e., for the PCCTS variables), at least one control variable was significantly associated with the variable missing data, thus facilitating data recovery. For example, race/ethnicity was highly significant in predicting exposure to the age three PCCTS adversities, with non-Hispanic blacks more likely to be exposed.

AIM 2 – ANALYSES

The goal of Aim 2 was to *examine the relationship between multiple adverse experiences and cognitive outcomes among children in the FFCW Study*. Findings from the literature review in Aim 1 were used to inform the way in which adversities were modeled in the Aim 2 analysis, including the formation of cumulative adversity indices and separate adversity domains at each wave (these are described in more detail below in the section, *Formulating Adversity Indices and Domains*). This led to the formation of the following sub-aims and hypotheses:

- Aim 2.1: Evaluate the relationship between cumulative adversity exposure (at infancy and ages three, five and nine years) and cognitive outcomes at ages five and nine years.
 - *Hypothesis 2.1a*: A higher total number of adverse experiences at each age will predict lower cognitive scores (both concurrently and longitudinally).

- *Hypothesis 2.1b*: A higher total number of adverse experiences during infancy and at age three will directly predict lower cognitive scores at ages five and nine, even after controlling for adverse exposures at ages five and nine.
- Aim 2.2: Examine whether higher scores in specific adversity domains during infancy and ages three, five and nine years influence cognitive outcomes at ages five and nine years.
 - *Hypothesis 2.2a*: Higher adversity domain scores, specifically lack of safety, family instability and economic hardship at each age (i.e., infancy and three, five and nine years) will predict lower cognitive outcomes (both concurrently and longitudinally).
 - *Hypothesis 2.2b*: Higher adversity domain scores during infancy and at age three will directly predict lower cognitive scores at ages five and nine, even after controlling for adverse exposures at ages five and nine.
- Aim 2.3: Examine whether exposure to the different adversity domains at age three mediates the relation between exposure to the adversity domains during infancy and cognitive outcomes at ages five and nine. This analysis was purely exploratory. No hypotheses were made.

To address these aims, exploratory analysis was first conducted in Stata 13 and used to create appropriate measures for each adverse experience and the cognitive outcomes. Hypotheses were then tested with these constructs using path models in Mplus 7.3. Each of these steps is described in more detail below.

Descriptive Statistics

Descriptive analyses were conducted with Stata 13. Means and standard deviations for continuous variables, and frequencies for categorical or dichotomous variables are presented in Tables 2.5-2.7 (and described above under the description of the analytic sample). Demographics of the final sample were similar to other studies using a similar sample from the FFCW Study (27). As expected with a relatively disadvantaged sample, mean scores on all of the age five and age nine child cognitive assessments were at or just below the normed average.

Selecting and Constructing Cognitive Outcome Measures

The FFCW Study collected several different measures of cognitive outcomes in children at ages five and nine. These included the measures of cognitive ability and functioning used in the final analysis that are described in the measures section above (Leiter Sustained Attention, Leiter Lack of Impulsivity, and PPVT-III at age five, and the WISC-IV Digit Span and PPVT-III at age nine) as well as additional measures of achievement that were ultimately not used in the final analysis (including the Woodcock-Johnson Letter-Word Assessment at age five and the Woodcock-Johnson Passage Comprehension and Applied Problems at age nine). During preliminary analyses, latent variables of cognitive function at age five and age nine were constructed using all of the available cognitive outcomes for each age. However, several of the measures had unacceptably low loadings on these factors (particularly the Leiter measures at age five and the Digit Span measure at age nine). Therefore, a decision was made to use only the individual manifest variables for measures of cognitive outcomes. Furthermore,

cognitive outcome variables were limited to those that captured a basic cognitive ability or function (i.e. the Leiter measures, PPVT-III and Digit Span). The Woodcock-Johnson measures, which are known to be measures of achievement and more likely to be influenced by factors not addressed in this study (e.g., quality of education) were excluded from further analysis. All cognitive outcomes were normally distributed. Z-scores ($M=0$, $SD=1$) were created for all cognitive outcomes and used in all analyses (aside from descriptive analyses) in order to facilitate comparisons across outcomes.

Correlations Among Adversities

Relationships among adversities within waves and across waves were examined using tetrachoric correlations for dichotomized variables and are shown in Table 2.9. Correlations are classified as low (<0.1), modest ($0.1-0.3$), moderate ($0.3-0.5$) and strong (>0.5). *Within waves*, strong correlations were observed at infancy between food and housing insecurity; at age three between severe psychological aggression and severe corporal punishment; at age five between food and housing insecurity; and at age nine between severe psychological aggression and severe corporal punishment, and between food and housing insecurity. Moderate to strong correlations for like adversities *across waves* were observed for nearly all adversities, suggesting some stability in exposure over time. The strongest correlations across time were observed for poverty, father incarceration, and exposure to community violence.

Formulating Cumulative Adversity Indices and Adversity Domains

In addition to the individual adversity measures described in the measures section above (e.g., severe physical abuse, severe psychological abuse, neglect), two other formulations of adversity exposures were constructed. First, a single cumulative index was created for each age. As described in Chapter 1, a cumulative index is a common way of measuring multiple adverse exposures. This measure is constructed by summing each individual exposure for each age into a single aggregate measure and is useful for evaluating the effects of dosage of adversity exposure. During infancy, eight adverse experiences were summed into a single cumulative index with a possible score ranging from 0-8. Eleven adversities were measured at years three, five and nine. A cumulative index for each of these ages was created, with total possible scores ranging from 0-11. Each adversity in the index was given equal weight. Mean cumulative index scores for this sample were 1.4 (SD=1.6) for infancy, 2.1 (SD=3.0) at age three, 2.2 (SD=3.1) at age five, and 2.3 (SD=3.0) at age nine. Correlations among cumulative indices across waves ranged from moderate ($r=0.37$ between infancy and age nine) to high ($r=0.53$ between age five and age nine, and $r=0.54$ between age three and age five).

Adversity domains are another useful method for studying multiple adverse exposures (described in more detail in Chapters 3 and 4). Domains provide information about the relative salience of particular types of adversities. In short, a domain-based approach groups adversities of a similar type either by a theoretically determined cumulative index score (i.e., food insecurity, housing insecurity and poverty level combined to create one cumulative score for *economic hardship*) or by empirical factor analytics.

In order to inform the formation of adversity domains, exploratory factor analysis (using principal components and maximum likelihood estimation methods and varimax rotation) was used to determine how the different adversities were related to one another and whether a common factor or set of factors explained shared variance across adversities within waves. Adversities were considered to load onto a factor if the factor loadings were greater than 0.4 and the uniqueness (or variability of the adversity not explained by the factor) was less than 0.6 (28).

The factor structure was first examined for groups of adversities within each wave hypothesized to be in the same domain (based on findings from the literature review, described in Chapter 3). For example, the *lack of safety* domain was hypothesized to include exposure to psychological aggression, physical abuse, neglect, exposure to community violence and domestic violence. The *instability* domain was hypothesized to include exposure to parental relationship instability, maternal depression, and father incarceration. The *economic hardship* domain was hypothesized to include living below the poverty level, food and housing instability. Then the factor structure for all adversities within each wave was examined. While the results of the exploratory factor analysis provided some evidence that the adversities loaded onto factors that corresponded with the hypothesized domains, the results were also problematic. In some cases, low loadings and dual-factor loadings were observed. Additionally, the factor structure did not hold across waves.

At this point, a decision was made to use the theoretically determined adversity domains rather than the empirically derived factors based on the recognition that for robust factors to be identified, children must experience the occurrence of multiple

adversities within a domain, which does not necessarily happen and is not a requirement for a theoretically defined domain. Theoretically determined domains, on the other hand, were more appropriate for capturing types of exposures that created a certain context, such as lack of safety, family instability or economic hardship. The *lack of safety* domain was intended to capture the number of exposures that threatened a child's safety, and therefore, this domain was created by summing the total number of exposures that may create this context, including severe psychological aggression, severe corporal punishment, neglect, community violence and domestic violence. Similar aggregate scores were created for and *family instability* domain and *economic hardship*. Table 2.10 lists the adversities included in each domain. Mean scores on the adversity domains ranged from 0.2-1.3 (SD=0.2-1.4) across waves for the lack of safety domain, 0.3-0.6 (SD=0.3-0.5) for the instability domain, and 0.6-0.7 (SD=0.5-0.6) for the poverty domain.

Bivariate Relationships

Bivariate relationships were then assessed between each cognitive outcome and all other variables (including all covariates, individual adversities, cumulative adversity indices for each age, and adversity domains for each age). Bivariate relationships were assessed both graphically (to assess linear relationships between the cumulative indices and all cognitive outcomes) and empirically (using univariate path models in Mplus 7.3).

Selection of Covariates and Adjusted Models

The final set of covariates used in the adjusted analysis included race, sex, maternal education and neonatal risk. Neonatal risk was a constructed variable defined as

born with low birth weight *or* as a twin. Twin and low birth weight was combined because the FFCW Study coded all children born as a twin as missing in birth weight. When examined as separate variables, these variables co-varied. Since twins are more likely to be born low birth weight, the two variables were combined into a single measure. Prenatal substance abuse variables (drinking, smoking, and drug use) were not included as covariates in the adjusted models because they were not significantly related to any of the cognitive outcomes in either the bivariate or multivariate analyses. Maternal cognitive ability was also dropped from the final analysis because this variable was missing 7% of responses (given that it was assessed at year 3, unlike all other covariates which were assessed at baseline), and this was problematic for using FIML.⁹ However, maternal cognitive ability was moderately correlated with maternal education ($r = 0.37$); therefore, maternal education was used as a proxy for maternal cognitive ability. Sensitivity analyses comparing models including maternal education and maternal cognitive ability showed no substantive differences in the relationships between adversities and cognitive outcomes between these models and those that did not control for mother cognitive ability.

After selecting the final group of controls, each cognitive outcome was regressed on each adversity from each wave, adjusting for controls. These models were useful for evaluating the individual relationships of each adversity on the cognitive outcomes, prior to conducting analyses with the cumulative indices and domains.

⁹ Exogenous variables are typically not recovered using FIML based on missing data theory.

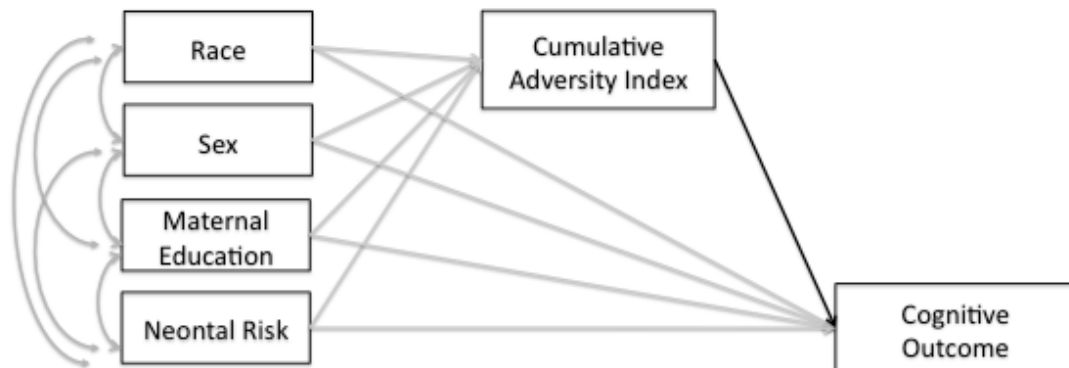
Path Analysis & Hypothesis Testing

Path analysis was conducted in Mplus Version 7.3 using FIML to test the stated hypotheses for Aim 2. Path analysis is a type of structural equation modeling that estimates a system of equations in which all variables are observed. Unlike latent variable models, path models assume perfect measurement among observed variables. Variables that only predict other variables are referred to as exogenous variables, and variables that are predicted by other variables are referred to as endogenous variables. Path analysis distinguishes three types of effects between variables: a direct effect is the influence that one variable has on another that is not mediated by any other variable in the model; an indirect effect is the influence of one variable on another through mediation of at least one other variable; and the total effect is the sum of the direct and indirect effects. Path analysis is, therefore, a useful method for studying mediation (29).

Path models were first evaluated for overall model fit using two fit indices. The root mean squared error of approximation (RMSEA) is an absolute fit index that compares the analytic model to a fully saturated (perfect) model; RMSEA <0.08 indicates acceptable fit and <0.05 close fit. The comparative fit index (CFI) is a relative fit index that compares the analytic model to a null model with no observed relationships between variables; CFI >0.9 indicates acceptable fit and >0.95 close fit. Although a third, χ^2 statistic indicates the exact fit of the model (i.e. that the observed variance/covariance matrix equals the variance/covariance matrix of the model), this index is highly sensitive to sample size and generally not recommended for evaluating overall model fit. In addition to model fit statistics, models were also evaluated by the significance of path estimates and the explained variance among the endogenous variables (29,30).

To test Hypothesis 2.1a, that *a higher total number of adverse experiences at each age will predict lower cognitive scores*, simple path models were run in which each cognitive outcome was individually regressed on the cumulative adversity index for each age—one at a time, controlling for race, sex, maternal education, and neonatal risk (Figure 2.2). Both the outcome and the cumulative adversity index were regressed on to the set of controls, and controls were set to correlate with one another, as illustrated in Figure 2.2 below with the gray arrows. All models were a good fit according to fit statistics. The significance of the direct path between each cumulative adversity index and each cognitive outcome (illustrated by the black line in the figure below) was used to test the stated hypothesis.

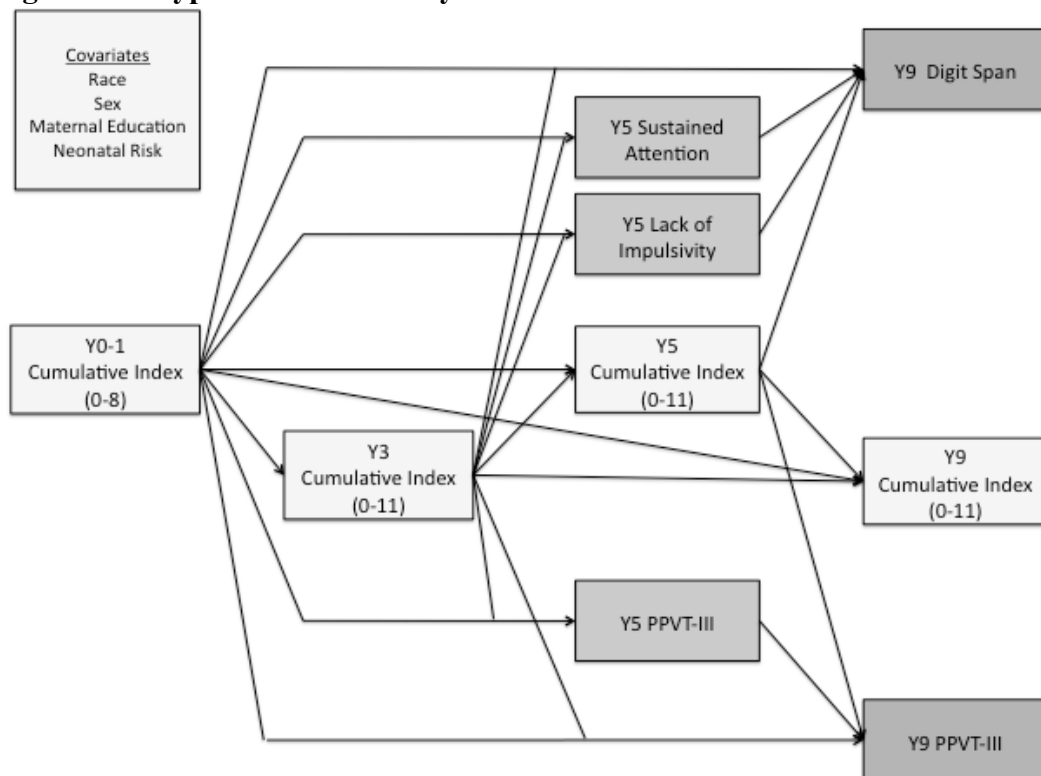
Figure 2.2. Hypothesis 2.1a Analytic Model



To test Hypothesis 2.1b, that *a higher total number of adverse experiences during infancy and at age three will directly predict lower cognitive scores at ages five and nine, even after controlling for adverse exposures at ages five and nine*, a full model was built that included all cognitive outcomes, and all cumulative adversity indices and covariates. The full model is shown in Figure 2.3 below, but the model was built in smaller steps, starting with all cognitive outcomes and covariates, then adding each cumulative

adversity index, one wave at a time. All models were a good fit according to fit statistics. The final model allowed for direct longitudinal paths between the cumulative adversity indices at each age and all subsequent cognitive outcomes (shown with black arrows) as well as direct longitudinal paths between each cumulative adversity index. Indicators within the same wave were correlated (not shown in the diagram). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model. The model was evaluated by the overall model fit, the significance of direct paths between each cumulative adversity index and each cognitive outcome, and the total variance explained in each cognitive outcome with the addition of each subsequent wave of cumulative adversity index.

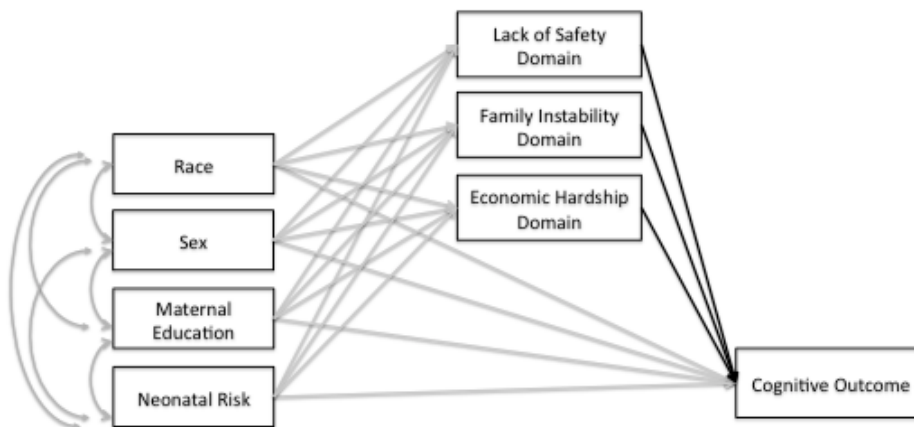
Figure 2.3. Hypothesis 2.1b Analytic Model ^a



^a Indicators within the same wave were correlated (not depicted in the diagram). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

To test Hypothesis 2.2a, that *higher adversity domain scores, specifically lack of safety, family instability and economic hardship at each age (i.e., infancy and ages three, five and nine years) predict lower cognitive outcomes*, simple path models were run, similar to those described 2.1a above, where each cognitive outcome was individually regressed on the set of adversity domains for each age – one wave at a time, controlling for race, sex, maternal education and neonatal risk (Figure 2.4). Both the outcome and the adversity domains were regressed on to the set of controls, and controls were set to correlate with one another, as illustrated in Figure 2.4 below with the grey arrows. The domains within each wave were also allowed to correlate with one another (arrows not shown). All models were a good fit according to fit statistics. The significance of the direct paths between each adversity domain and each cognitive outcome (illustrated by the black lines in the figure below) were used to test the stated hypothesis.

Figure 2.4. Hypothesis 2.2a Analytic Model



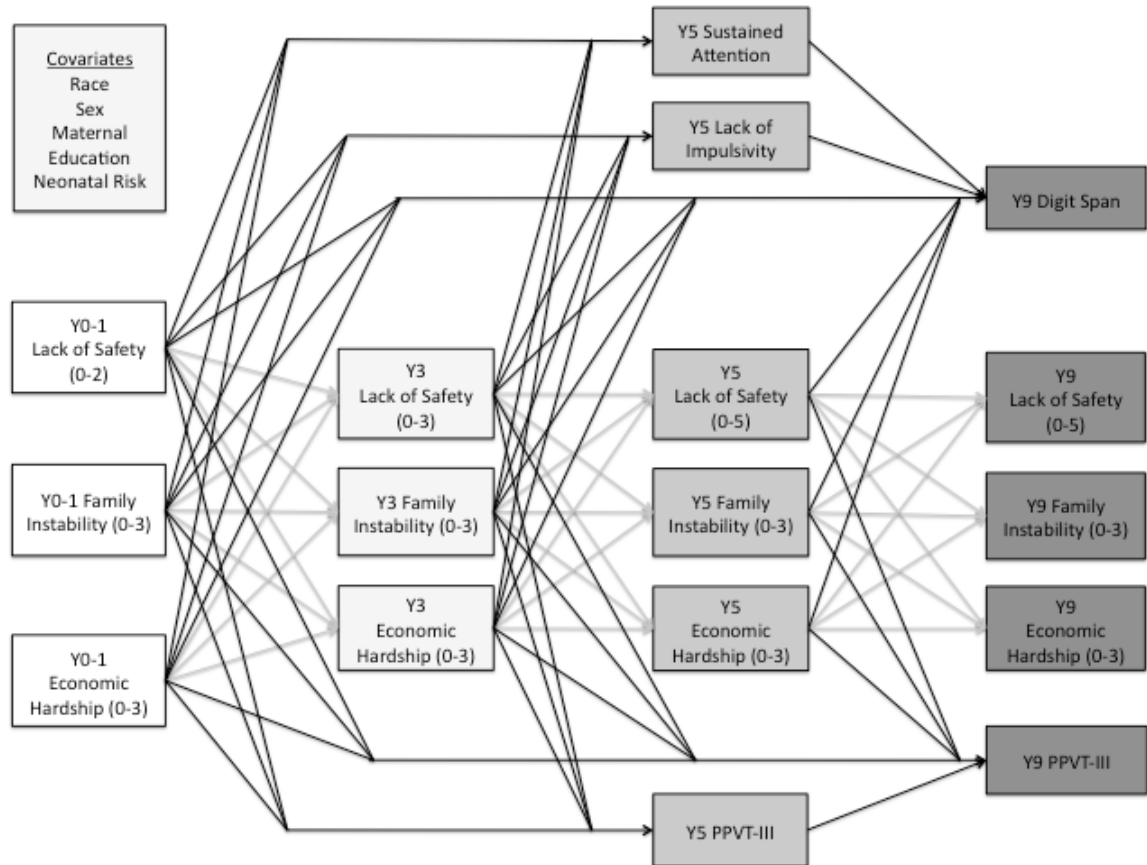
To test Hypothesis 2.2b that *higher adversity domain scores during infancy and at age three directly predict lower cognitive scores at ages five and nine, even after controlling for adverse exposures at ages five and nine*, a full model was built that included all cognitive outcomes, and all adversity domains and covariates. The full

model is shown in Figure 2.5 below, but again, the model was built in smaller steps, starting with all cognitive outcomes and covariates, then adding each wave of adversity domains, one wave at a time. All models were a good fit according to fit statistics. The final model allowed for direct longitudinal paths between the adversity domains at each age and all subsequent cognitive outcomes (shown with the black arrows) as well as direct longitudinal paths between all adversity domains (paths between one lag shown with the grey arrows; paths greater than one lag are not shown). Indicators within the same wave were correlated (not shown in the diagram). The full model controlled for race, sex, maternal education and neonatal risk – each covariate was correlated with one another and pointed to all other variables in the model. The model was evaluated by the overall model fit, the significance of direct paths between each adversity domain and each cognitive outcome, and the total variance explained in each cognitive outcome with the addition of each subsequent wave of adversity domains.

To examine Aim 2.3, *whether exposure to the different adversity domains at age three mediates the relation between exposure to the adversity domains during infancy and cognitive outcomes at ages five and nine*, specific indirect paths were tested.

According to MacKinnon and colleagues, for mediation to be present, the direct paths to and from the mediating variables had to both be significant (31). In this case, significant direct paths from an infancy domain to an age three domain and from the age three domain to the cognitive outcome must both be present to indicate mediation. This method does not depend on the attenuation of the direct effect from the infancy domain to the cognitive outcome, after accounting for the age three domain, as suggested in alternative methods of mediation by Baron and Kenny (32).

Figure 2.5. Hypothesis 2.2b Analytic Model ^a



^a The conceptual model allowed for direct longitudinal paths between all adversity domains (paths representing one lag shown with the grey arrows; paths greater than one lag are not shown). All indicators within the same wave were correlated (not depicted in the diagram). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

AIM 3 – ANALYSES

The goal of Aim 3 was to build upon the findings in Aim 2 to *identify factors that mediated and moderated the relationship between multiple adversity exposures and cognitive outcomes among children in the FFCW Study*. The specific sub-aims and hypotheses were as follows:

- Aim 3.1: Examine whether characteristics of the home environment, including maternal warmth and availability of reading materials, mediate the

relationship between adversity domains (at infancy and age three) and cognitive outcomes (at ages five and nine).

- *Hypothesis 3.1a*: Availability of reading materials mediates the relation between the economic hardship domain and cognitive outcomes.
- *Hypothesis 3.1b*: Maternal warmth mediates the relation between the adversity domains and cognitive outcomes.
- Aim 3.2: Examine whether gender moderates the mediation process above.

This analysis was purely exploratory. No hypotheses were made.

Preliminary Analyses

Means and standard deviations for continuous variables, frequencies for categorical or dichotomous variables, correlations between study variables, and univariate path models regressing each cognitive outcome on the key study variables were examined using in Mplus 7.3 (33). Linear assumptions between the adversity domains, mediating variables and cognitive outcomes were examined graphically in Stata 13.

Hypothesis Testing

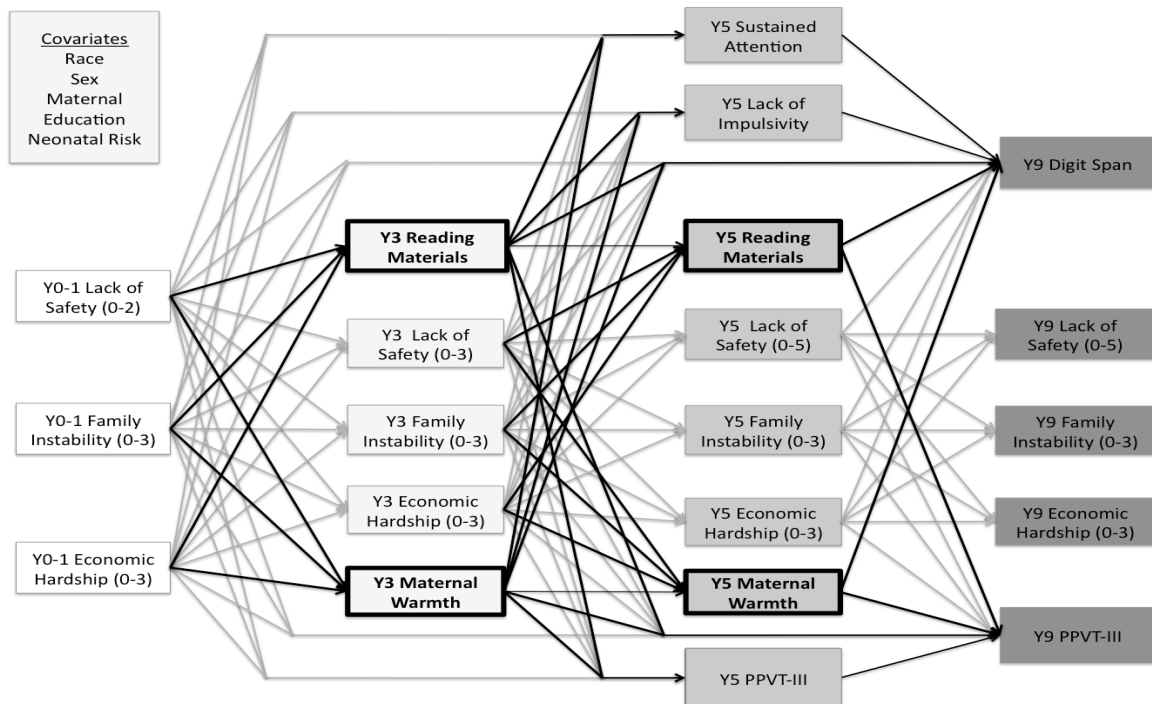
As described in the description of study measures above, two variables from the HOME scale observations at ages three and five were hypothesized to mediate the relation between the adversity domains and cognitive outcomes: availability of reading materials (measured by the number of child and adult books in the house) and maternal

warmth (measured by observed interactions between the mother and child). To test the hypotheses that *availability of reading materials mediates the relationship between the economic hardship domain and cognitive outcomes, and that maternal warmth mediates the relationship between all adversity domains and cognitive outcomes*, an additional path model was estimated (shown Figure 2.6 below). This model built upon Figure 2.5 from Aim 2 (all paths from the Aim 2 model are shown with grey arrows). The mediating variables at ages three and five are shown in boxes with a bold outline, and the new paths that were included in the model are shown with black arrows. Mediation was tested by evaluating the significance of the indirect paths from the infancy domains to the age five and nine cognitive outcomes through the age three mediating variables, and from the age three domains to the age nine cognitive outcomes through the age five mediating variables. According to MacKinnon and colleagues, for mediation to be present, both paths from the domain to the mediating variable and from the mediating variable to the cognitive outcome had to be significant (31,34). Mediation was tested using the Delta Method, which, similar to the Sobel Test computes a Z-score based on the product-of-coefficients of the indirect effects (34).

To examine whether *gender moderates the mediation process* (from the sub-aim above), multigroup analysis, stratifying the model by gender, was used to compare the regression coefficients between males and females. A fully constrained model where all paths were constrained to be equal for males and females was compared to a model where all paths were estimated freely. A significant change in chi-square between the two models was considered evidence of moderation. The significance of the indirect path estimates described above were then examined to identify specific differences by gender.

Multi-group analysis was selected to test for moderation (rather than the use of interaction terms) for conceptual clarity, given the complexity of the overall model.

Figure 2.6. Hypothesis 3.1a and 3.1b Analytic Model ^a



^aThe conceptual model allowed for direct longitudinal paths between all adversity domains (paths representing one lag shown with the grey arrows; paths greater than one lag are not shown). All indicators within the same wave were correlated (not depicted in the diagram). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

STRENGTHS AND LIMITATIONS OF QUANTITATIVE METHODS

There are several strengths to these methods. This study draws upon a rich set of reliable and valid measures in a large, longitudinal sample that allows for studying the temporal influence of adverse exposures. An advantage of path analysis is the ability to model complex relationships among multiple dependent and independent variables (35). This feature is useful for testing broader theories about the relations between variables. An additional advantage of path analysis is that it decomposes correlations among variables into both direct and indirect pathways, which is particularly useful in tests of

mediation (29,35). Furthermore, the statistical software used to run these analyses also allows for more robust methods for handling missing data (25). FIML produces unbiased results, even with a high percentage of MAR or MCAR data (25).

These methods also present several limitations. The measures of adversity are obtained from interviews with biological mothers who may have poor recall or provide socially desirable responses. Therefore, it is likely that these data underestimate the actual occurrence of adversity in the study sample. With respect to timing of exposures, two issues arise. First, not all adversity measures assess exposure over the same time frame. While most adversity measures assess exposure over the last year, domestic violence and relationship stability assess exposure since the previous wave. Additionally, the exact ages of children varied for each wave. Therefore, the study conclusions about temporality can only be generalized to early, middle, and late childhood rather than specific ages.

Table 2.1. Scoring Criteria for Literature Review

Criteria	Points
Representativeness	<ul style="list-style-type: none">• Sample not representative of the target population (1 point)• Sample moderately representative of target population (2 points)• Sample representative of the target population (3 points)
Study Design	<ul style="list-style-type: none">• Cross-sectional, case study, case control (1 point)• Retrospective cohort (2 point)• Prospective cohort/longitudinal (3 points)
Sample Size	<ul style="list-style-type: none">• <100 (1 point), 100-500 (2 points), >500 (3 points)
Data Collection Methods	<ul style="list-style-type: none">• Data collection tools are neither valid nor reliable (1 point)• Data collection tools are valid but not reliable (2 points)• Data collection tools are valid and reliable (3 points)
Confounders	<ul style="list-style-type: none">• No confounders were accounted for (1 point)• Some confounders were accounted for (2 points)• Most confounders were accounted for (3 points)
Analyses	<ul style="list-style-type: none">• Correlations, descriptive (1 point)• Simple regression, bivariate/ANOVA (2 points)• ANCOVA, multivariate regression, structural equation modeling (3 points)
Adversity Assessment	<ul style="list-style-type: none">• Accounts for occurrence only (1 point)• Accounts for severity OR duration (2 points)• Accounts for severity and duration (3 points)

Table 2.2. Number of Studies for Each Cognitive Outcome by Study Design

Cognitive Outcomes	Case-Control	Cross- Sectional	Longitudinal
General Cognitive Ability	1	5	11
Executive Function	0	2	5

Table 2.3. Summary of Measures

Variable	Measure	Response Coding	Source
Cognitive Outcomes			
Sustained Attention	Leiter International Performance Scale —Revised: assesses children’s ability to maintain attention to a specific stimulus and to suppress their impulses. The number of correct responses reflects the child’s sustained attention.	Continuous score – standardized (M=10, SD=3, $\alpha = 0.83$)	Y5 In-Home Assessment
Lack of Impulsivity	Leiter International Performance Scale —Revised: assesses children’s ability to maintain attention to a specific stimulus and to suppress their impulses. The number of incorrect responses, reverse coded, reflects the lack of impulsivity.	Continuous score – standardized (M=10, SD=3, $\alpha = 0.83$)	Y5 In-Home Assessment
<i>PPVT-III</i>	Peabody Picture Vocabulary Test (PPVT-III): measures receptive vocabulary and screens for verbal ability. The interviewer reads a word and asks the child to identify the picture in the easel that corresponds to that word.	Continuous score – standardized (M=100, SD=15, $\alpha = 0.93$)	Y5, Y9 In-Home Assessment
<i>Digit Span</i>	16-item Digit Span subtest of the Wechsler Intelligence Scale for Children (WISC- IV Digit Span): assesses short-term memory, sequencing skills, attention and concentration. Children are verbally given a sequence of numbers and asked to repeat the numbers back either forward or backwards.	Continuous score – standardized (M=10, SD=3, $\alpha = 0.92$)	Y9 In-Home Assessment
Adverse Experiences			
<i>Severe Psychological Aggression</i>	Mothers reported on three items from the Psychological Aggression Subscale of the Parent-Child Conflict Tactics Scales (PCCTS): severe psychological aggression defined as one or more occurrence of any item (swearing or cursing at child, calling child dumb or lazy, or threatening to kick child out of the house.) ¹⁰	0 = no severe psychological aggression the past year 1 = at least one occurrence of severe psychological aggression in the past year	Y3, Y5, Y9 PCG Survey
<i>Severe Corporal Punishment</i>	Mothers reported on three items from the Physical Assault Subscale of the PCCTS: severe corporal punishment defined as one or more occurrence of any item (hit with hard object, pushed, or shook the child) in last year. ¹¹	0 = no severe corporal punishment in the past year 1 = at least one occurrence of severe corporal punishment in past year	Y3, Y5, Y9 PCG Survey
<i>Child Neglect</i>	Mothers reported on the five-item Neglect Subscale of the PCCTS: neglect defined as one or more occurrence of any item (left child alone, not able to show love, did not provide food, did not provide medical care, drugs or alcohol got in the way). ²	0 = no neglect in the past year 1 = at least one occurrence of neglect in past year	Y3, Y5, Y9 PCG Survey

¹⁰ Scoring described in Straus and Field, 2003, “Psychological Aggression by American Parents.”

¹¹ Scoring described in 3&5 Year In-Home User Guides for FFCW Study. Cut-offs described in <http://pubpages.unh.edu/~mas2/CTS28.pdf>.

Table 2.3. Summary of Measures (continued)

Variable	Measure	Response Coding	Source
<i>Intimate Partner Violence (IPV)</i>	Mothers reported on three validated questions about whether they had been slapped or kicked, hit with a fist or hard object, or forced to have sex or do something sexual by a current partner (father or other). Mothers were also asked whether they had been in a physical fight with the father or a current partner in front of the child since the last interview . IPV defined as a positive response to any of these questions.	0 = no mother report of IPV since last wave 1 = mother reports IPV since last wave	Y1, Y3, Y5, Y9 Core Mother
<i>Exposure to Community Violence</i>	At baseline, mothers reported on how safe their streets were at night (very safe, safe, unsafe, very unsafe). At ages 3, 5, and 9, mothers reported on three questions: if they saw someone get hit by someone else, if they saw someone get attacked with a weapon, and if they saw someone get shot.	0 = safe or very safe (baseline) or no exposure in the last year (Y3-Y9) 1 = unsafe or very unsafe streets (baseline) or at least one exposure in last year (Y3-Y9)	Y0 Core Survey; Y3, Y5, Y9 PCG Survey
<i>Parental Relationship Instability</i>	Mothers reported whether: they were married, cohabitating or single. Stability was defined as having the same parent structure since the previous wave or moving from a cohabitating relationship to a married relationship since the previous wave. Moving from a married relationship to a cohabitating or single status, or moving from cohabitating relationship to single status was categorized as unstable.	<i>Infancy</i> 0 = 2 parents (married or cohabitating) 1 = 1 parent (single) Y3, Y5, Y9 0 = 1 or 2 parent stable since previous wave 1 = 1 or 2 parent unstable since previous wave	Y0, Y3, Y5, Y9 Core Mother
<i>Maternal Depression</i>	Mothers reported on the Composite International Diagnostic Interview – Short Form (CIDI-SF): a subset of items from the CIDI-SF used to classify a probable case for a depressive episode in the past year. Probable cases are defined by pre-determined criteria.	0 = mother not a probable case for a depressive episode in the past year 1 = mother a probable case for depressive episode in the past year	Y1, Y3, Y5, Y9 Core Mother
<i>Parental Incarceration</i>	Biological mothers and fathers were asked whether the father was currently in jail or prison. Responses from both mothers and fathers combined into a single measure for each wave. Responses for the baseline and Year 1 waves were combined.	0 = father not currently in jail 1 = father currently in jail	Y0, Y1, Y3, Y5, Y9 Core Mother/Father
<i>Income to Needs</i>	Mother report of family income adjusted by the number of adults and children in the household, using the official poverty thresholds.	0 = Above the poverty line at wave 1 = Below the poverty line at wave	Y0, Y3, Y5, Y9 Core Mother
<i>Housing Insecurity</i>	Mothers were asked whether they had been evicted from their home in the past 12 months, stayed in a shelter/car or abandoned vehicle, did not pay full rent or mortgage, or if they had moved in with other people because of financial problems. Mothers responding “yes” to at least one of these questions were categorized as experiencing housing insecurity.	0 = no housing insecurity in the last year 1 = housing insecurity in the last year	Y1, Y3, Y5, Y9 Core Mother
<i>Food Insecurity</i>	Mothers were asked whether, in the past 12 months, they were ever hungry but could not afford to buy more food. Mothers responding “yes” to question were characterized as experiencing food insecurity.	0 = no food insecurity in the last year 1 = food insecurity in the last year	Y3 PCG; Y1, Y5, Y9 Core Mother

Table 2.3. Summary of Measures (continued)

Variable	Measure	Response Coding	Source
Mediators			
<i>Maternal Warmth</i>	Interviewers observed five (yes/no) items from the HOME observation parental warmth subscale: parent vocalized to child, parent responded verbally to child's vocalizations, parent praised child twice, parent conveys positive feelings, parent caressed or kissed child.	Responses summed to create a continuous score for each wave	Y3, Y5 In-Home Assessment
<i>Availability of Reading Materials</i>	Interviewers observed two items from age 3 HOME observation subscale: the number of adult books in the house, and the number of books for kids in the house; the age 5 HOME observation consisted of two items: number of books to learn the alphabet and number of books in the house.	Responses summed to create a continuous score for each wave	Y3, Y5 In-Home Assessment
Covariates			
<i>Birth Weight</i>	Mother report of baby birth weight.	0 = normal birth weight (>2500 g) 1 = low birth weight (≤ 2500 g)	Y0 Core Mother
<i>Singleton/Twin</i>	Constructed variable indicating whether singleton or twin birth.	0 = singleton 1 = twin	Y0 Core Mother
<i>Prenatal Alcohol Use</i>	Mothers asked how often they drank alcoholic beverages during pregnancy.	0 = less than several times/month 1 = several times/month or more	Y0 Core Mother
<i>Prenatal Drug Use</i>	Mothers asked how often they did drugs (i.e. marijuana, crack, heroine, or cocaine) during pregnancy.	0 = no use 1 = any drug use	Y0 Core Mother
<i>Prenatal Smoking</i>	Mothers asked how often they smoked cigarettes during pregnancy.	0 = no smoking 1 = any smoking	Y0 Core Mother
<i>Parental Cognitive Ability</i>	Wechsler Adult Intelligence Scale – Revised (WAIS-R) Similarities subtest: includes eight items that measure verbal concept formation and reasoning abilities, though the items may also reflect long-term memory and cultural opportunities.	Continuous score - standardized	Y3 Core Mother
<i>Maternal Education</i>	Mothers asked about their highest level of schooling completed, ranging from no formal school to graduate or professional school.	1 = less than high school 3 = some college 2 = high school 4 = college	Y0 Core Mother
<i>Child Sex</i>	Mother report of child sex.	0 = male 1 = female	Y0 Core Mother
<i>Child Race</i>	Mother report of race/ethnicity.	1 = white 3 = Hispanic 2 = black, non-Hispanic 4 = other	Y0 Core Mother
<i>Maternal Age at Birth</i>	Mother report of age.	Continuous, in years	Y0 Core Mother

Table 2.4. Adversity Measures Collected at Each Wave

	Baseline ^a	Year 1 ^a	Year 3	Year 5	Year 9
Severe Psychological Aggression			X	X	X
Severe Corporal Punishment			X	X	X
Neglect			X	X	X
Maternal Depression		X	X	X	X
Intimate Partner Violence		X	X	X	X
Relationship Instability ^b	X		X	X	X
Father Incarceration	X	X	X	X	X
Community Violence ^b	X		X	X	X
Below Poverty Level	X		X	X	X
Housing Insecurity		X	X	X	X
Food Insecurity		X	X	X	X

^aMeasures from Baseline and Year 1 were combined to form a single wave, referred to as *infancy*.

^bThe measure used at baseline was different from the measure used in years 3, 5, and 9.

Table 2.5. Original and Final Analytic Sample (Covariates and Cognitive Outcomes)

	FFCW Sample (N=4657)		Excluded (N=1681)		Final Sample (N=2976)			p<0.05
	N	% Yes	N	% Yes	N	% Yes	% Missing	
<i>Maternal Education at Child's Birth</i>							0.1	
<High School	1631	35.1	685	40.8	946	31.8		***
HS or Equivalent	1408	30.3	469	27.9	939	31.6		**
Some College	1118	24.0	354	21.1	764	25.7		***
College or Grad	495	10.6	172	10.2	323	10.9		
<i>Mother Race</i>							0.2	
White, non-Hispanic	971	20.9	380	22.7	591	19.9		*
Black, non-Hispanic	2213	47.6	719	42.9	1494	50.3		***
Hispanic	1273	27.4	489	29.2	784	26.4		*
Other	189	4.1	89	5.3	100	3.4		***
<i>Female Child</i>	2237	48.0	809	48.1	1428	48.0	0	
<i>Birth Weight <2500g</i>	450	9.9	191	11.7	259	8.9	2.7	**
<i>Twins</i>	85	1.8	27	1.6	58	2.0	0	
<i>Prenatal Alcohol Use ≥1x/mo</i>	116	2.5	61	3.6	55	1.9	0.2	***
<i>Any Prenatal Drug Use</i>	260	5.6	153	9.1	107	3.6	0.2	***
<i>Any Prenatal Smoking</i>	898	19.3	391	23.4	507	17.1	0.1	***
	Mean	sd	Mean	sd	Mean	sd	% Missing	
<i>Mean Maternal Age</i>	25.2	6.0	25.5	6.1	25.1	6.0	<0.1	*
<i>Maternal WAIS-R Score</i>	6.7	2.7	6.5	2.7	6.8	2.7	6.9	***
<i>Child Age (in months) Y3 Core</i>	35.8	2.6	36.3	2.8	35.6	2.4	6.6	***
<i>Child Age (in mo) Y5 Core</i>	61.9	2.9	62.7	3.1	61.6	2.7	5.7	***
<i>Child Age (in mo) Y5 In-Home</i>	63.8	2.9	64.4	3.0	63.7	3.1	38.0	***
<i>Child Age (in mo) Y9 Core</i>	112.7	4.6	114.3	5.1	112.4	4.4	0.9	***
<i>Child Age (in mo) Y9 In-Home</i>	111.7	4.8	112.1	4.7	111.6	4.8	0.1	
<i>Y5 Cognitive Outcomes</i>								
Leiter Sustained Attn.	12.8	3.3	12.5	3.4	12.9	3.3	44.3	*
Leiter Impulse Control	10.1	2.8	10.2	2.8	10.1	2.9	44.3	
PPVT -III	93.4	15.8	89.7	16.8	94.3	15.4	38.1	***
<i>Y9 Cognitive Outcomes</i>								
WISC Digit Span	9.4	2.8	9.3	2.8	9.4	2.8	0.2	
PPVT-III	93.0	14.8	92.1	13.7	93.1	14.9	0.5	

FFCW Sample: Starts with baseline 20-city sample and excludes ineligible. **Final Sample:** Starts with 20-city sample, excludes ineligible, includes all families with at least one Y9 outcome, and includes only cases where moms completed PCG survey.

Significance tests are based on chi-squared tests for categorical variables and t-tests for continuous variables. Tests pertain to differences between those included in the final analytical sample, versus not.

*** P<0.001; ** p<0.01; * p<0.05

Table 2.6. Original and Final Analytic Sample (Adverse Experiences)

Table 2: Original and Final Family Experiences								
	FFCW Sample (N=4657)		Excluded (N=1681)		Final Sample (N=2976)			p<0.05
	N	% Yes	N	% Yes	N	% Yes	% Missing	
Severe Psych. Aggression								
Year 3	687	22.6	176	22.6	511	22.6	24.0	
Year 5	886	31.2	184	29.0	702	31.9	26.0	
Year 9	1353	42.4	132	42.0	1221	42.4	3.3	
Severe Corp. Punishment								
Year 3	927	30.6	214	27.3	713	31.7	24.4	*
Year 5	1093	38.7	237	37.3	856	39.1	26.4	
Year 9	1272	41.1	111	37.5	1161	41.5	6.0	
Exposed to Neglect								
Year 3	330	10.8	103	13.1	227	10.0	23.8	*
Year 5	311	11.0	79	12.3	232	10.6	26.4	
Year 9	699	21.8	59	19.0	640	22.1	2.7	
Maternal Depression								
Year 1	489	11.8	170	12.7	319	11.4	5.7	
Year 3	573	14.3	176	14.3	397	14.3	6.7	
Year 5	457	11.7	142	12.8	315	11.2	5.8	
Year 9	417	12.4	77	16.8	340	11.7	2.1	**
Intimate Partner Violence								
Year 1	296	7.2	103	7.8	193	7.0	6.7	
Year 3	229	5.7	86	7.0	143	5.2	6.7	*
Year 5	281	7.2	84	7.5	197	7.1	5.8	
Year 9	220	6.5	29	6.26	191	6.5	1.3	
Relationship Instability								
Year 1	1836	39.4	648	38.6	1188	39.9	<1.0	
Year 3	711	17.7	230	18.6	481	17.3	6.8	
Year 5	413	11.1	132	12.9	281	10.5	9.7	*
Year 9	311	9.8	46	11.3	265	9.6	6.8	
Father Currently in Jail								
Year 1	355	8.7	132	9.8	223	8.2	8.7	
Year 3	325	8.3	97	7.8	228	8.5	9.6	
Year 5	318	8.4	89	7.9	229	8.6	10.6	
Year 9	233	6.4	30	4.5	203	6.8	0.2	*
Community Violence								
Year 1	786	17.0	326	19.5	460	15.5	<1.0	***
Year 3	1120	36.7	295	37.7	825	36.4	23.9	
Year 5	932	33.0	219	34.3	713	32.6	26.4	
Year 9	861	24.5	111	20.0	750	25.4	0.6	**
Below Poverty Level								
Year 1	1699	36.3	652	38.8	1038	34.9	0.0	**
Year 3	1683	41.9	546	44.2	1137	40.9	6.7	
Year 5	1612	41.1	502	44.9	1110	39.5	5.7	**
Year 9	1257	37.2	177	39.3	1080	36.8	1.4	
Housing Insecurity								
Year 1	902	21.8	295	22.1	607	21.7	5.8	
Year 3	805	20.1	267	21.6	538	19.2	6.8	
Year 5	797	20.4	239	21.5	558	19.9	5.9	
Year 9	861	25.3	136	29.4	725	24.6	1.0	*
Food Insecurity								
Year 1	188	4.5	66	4.9	122	4.3	5.7	
Year 3	121	4.0	33	4.3	88	3.9	25.0	
Year 5	257	6.5	78	7.0	179	6.4	5.8	
Year 9	242	7.1	44	9.5	198	6.7	1.0	*

*** p<0.001; ** p<0.01; * p<0.05; Significance tests are based on chi-square tests for categorical variables and t-tests for continuous variables. Tests pertain to differences between those included in the final analytical sample, versus not.

Table 2.7. Original and Final Analytic Sample (Mediating Variables)

	FFCW Sample (N=4657)		Excluded (N=1681)		Final Sample (N=2976)			p<0.05
	N	% Yes	N	% Yes	N	% Yes	% Missing	
<i>Year 3 Parental Warmth</i>								
Parent vocalized to child	1801	90.3	386	87.3	1415	91.1	47.8	*
Parent responded verbally to child vocalizations	1850	92.7	406	91.9	1444	93.0	47.8	
Parent praised child twice	1646	82.9	357	80.4	1289	83.7	48.2	
Parent conveys positive feelings	1857	93.9	404	91.4	1453	94.7	48.4	*
Parent caressed or kissed child	1562	78.7	331	74.9	1231	79.7	48.1	*
<i>Year 5 Parental Warmth</i>								
Parent vocalized to child	1786	88.2	317	86.4	1469	88.6	44.3	
Parent responded verbally to child vocalizations	1825	90.6	325	89.0	1500	90.9	44.6	
Parent praised child twice	1389	68.8	241	65.7	1148	69.5	44.5	
Parent conveys positive feelings	1887	93.2	340	92.1	1547	93.4	44.4	
Parent caressed or kissed child	1186	58.9	210	57.2	976	59.3	44.7	
	Mean	sd	Mean	sd	Mean	sd	% Missing	
<i>Year 3 Availability of Reading Materials</i>								
# of adult books in house (ordinal)	1.8	1.1	1.7	1.1	1.8	1.0	24.0	
# of books for kids in house (ordinal)	3.9	0.5	3.8	0.5	3.9	0.5	23.8	*
<i>Year 5 Availability of Reading Materials</i>								
# books to learn alphabet (ordinal)	3.4	0.9	3.2	0.9	3.4	0.9	25.9	*
# books in house (ordinal)	3.7	0.6	3.7	0.7	3.7	0.6	26.0	**

Significance tests are based on chi-squared tests for categorical variables and t-tests for continuous variables. Tests pertain to differences between those included in the final analytical sample, versus not.

*** p<0.001; ** p<0.01; * p<0.05

Table 2.8. Sample Attrition (using final analytic sample)

	Baseline (Birth)	Year 1	Year 3	Year 5	Year 9
Core Mother Survey	2976 (100%)	2808 (94.4%)	2780 (93.4%)	2807 (94.3%)	2950 (99.1%)
In-Home PCG Survey			2273 (76.4%)	2213 (74.4%)	2972 (99.9%)
Home Observation			1596 (53.6%)	1665 (56.0%)	
Cognitive Assessment				1839 (60.0%)	2976 (100%)

Table 2.9. Correlations Among Individual Adversities

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)
(1) Y1 IPV	-																		
(2) Y1 ECV	.11	-																	
(3) Y1 Single	.06	.07	-																
(4) Y1 MD	.26	.12	.10	-															
(5) Y1 Jail	.26	.10	.37	.12	-														
(6) Y1 Poverty	.14	.24	.33	.08	.31	-													
(7) Y1 Housing	.29	.24	.11	.32	.13	.15	-												
(8) Y1 Food	.23	.23	.04	.36	.18	.20	.54	-											
(9) Y3 Psych Aggression	.20	.15	.08	.08	.15	.14	.08	.25	-										
(10) Y3 Corp Punishment	.09	.10	.14	.00	.06	.13	.07	.11	.52	-									
(11) Y3 Neglect	.22	.07	.10	.10	.17	.18	.19	.25	.35	.24	-								
(12) Y3 IPV	.57	.12	-.15	.29	.07	.12	.24	.15	.14	.09	.19	-							
(13) Y3 ECV	.14	.25	.26	.16	.30	.29	.20	.14	.29	.20	.25	.11	-						
(14) Y3 Relationship Instability	.28	.10	-.90	.04	.08	.07	.18	.11	.10	.09	.10	.39	.09	-					
(15) Y3 MD	.20	.15	.05	.53	.15	.06	.22	.30	.12	.11	.24	.30	.16	.13	-				
(16) Y3 Jail	.21	.10	.36	.08	.76	.30	.16	.06	.16	.13	.11	.19	.19	.22	.15	-			
(17) Y3 Poverty	.20	.24	.34	.10	.33	.62	.25	.22	.17	.11	.18	.14	.30	.19	.09	.31	-		
(18) Y3 Housing	.14	.17	.12	.25	.12	.10	.49	.31	.12	.18	.23	.21	.21	.16	.31	.17	.24	-	
(19) Y3 Food	.28	.13	.17	.27	.18	.14	.36	.54	.34	.17	.41	.20	.15	.09	.35	.12	.25	.34	-
(20) Y5 Psych Aggression	.23	.10	.14	.17	.13	.13	.18	.17	.52	.28	.23	.16	.24	.02	.10	.02	.14	.16	.18
(21) Y5 Corp Punishment	.11	.14	.15	.07	.16	.14	.14	.09	.29	.52	.22	.14	.16	.04	.11	.10	.15	.18	.10
(22) Y5 Neglect	.23	.10	.02	.19	.13	.06	.17	.14	.27	.06	.41	.17	.14	.02	.13	.11	.17	.15	.32
(23) Y5 IPV	.33	.07	-.02	.16	.06	.12	.19	.18	.16	.05	.18	.43	.13	.05	.25	.04	.10	.23	.17
(24) Y5 ECV	.08	.31	.25	.14	.19	.27	.18	.04	.25	.18	.22	.22	.59	.00	.12	.16	.30	.14	.09
(25) Y5 Relationship Instability	-.12	-.05	-.13	-.01	-.12	.06	-.07	-.12	.05	-.05	.02	.07	.15	-.67	.01	-.10	-.03	.03	.06
(26) Y5 MD	.23	-.07	-.04	.48	.17	.08	.21	.33	.14	.19	.20	.25	.14	.11	.58	.11	.05	.20	.27
(27) Y5 Jail	.12	.12	.36	.05	.68	.29	.17	.12	.20	.10	.20	.03	.23	.18	.13	.81	.30	.20	.17
(28) Y5 Poverty	.15	.27	.34	.13	.35	.57	.22	.20	.18	.06	.15	.18	.32	.10	.14	.29	.73	.21	.28
(29) Y5 Housing	.11	.18	.08	.21	.15	.10	.43	.30	.09	.14	.16	.26	.20	.12	.25	.15	.16	.50	.35
(30) Y5 Food	.27	.15	.13	.35	.22	.18	.34	.54	.12	.26	.22	.17	.15	.15	.34	.10	.25	.36	.55
(31) Y9 Psych Aggression	.16	.09	.06	.16	.07	.01	.12	.12	.39	.48	.18	.20	.17	.06	.17	-.12	.05	.11	.14
(32) Y9 Corp Punishment	.08	.07	.14	.14	.10	.09	.15	.13	.20	.20	.20	.11	.23	.03	.10	.06	.07	.13	.06
(33) Y9 Neglect	.15	.12	-.02	.18	.04	.10	.16	.28	.23	.04	.27	.17	.09	.07	.17	-.03	.01	.18	.27
(34) Y9 IPV	.33	.05	-.13	.08	-.02	.00	.06	.16	.05	.13	.12	.16	.05	-.07	.14	-.04	.03	.08	.22
(35) Y9 ECV	.07	.30	.24	.10	.21	.34	.11	.11	.14	-.06	.15	.13	.51	.00	.13	.16	.29	.14	.14
(36) Y9 Relationship Instability	.10	.11	-.15	.05	-.01	.02	-.03	.08	-.06	.11	-.10	-.10	.05	-.26	.03	-.14	-.03	.02	-.05
(37) Y9 MD	.10	.19	.08	.38	.04	.16	.10	.29	.08	.11	.15	.17	.20	.10	.50	.11	.08	.21	.30
(38) Y9 Jail	.12	.09	.29	.05	.55	.26	.15	.18	.16	.12	.15	.08	.20	.11	.02	.67	.26	.19	.10
(39) Y9 Poverty	.14	.21	.26	.17	.27	.52	.18	.2	.17	.10	.17	.20	.33	.12	.12	.24	.62	.26	.29
(40) Y9 Housing	.09	.14	.07	.10	.13	.14	.28	.23	.07	.10	.15	.15	.21	.15	.23	.13	.17	.36	.26
(41) Y9 Food	.21	.13	.06	.25	.16	.19	.25	.36	.05	.14	.16	.20	.20	.08	.26	.21	.18	.27	.46

Table 2.9. Correlations Among Individual Adversities (continued)

	(20)	(21)	(22)	(23)	(24)	(25)	(26)	(27)	(28)	(29)	(30)	(31)	(32)	(33)	(34)	(35)	(36)	(37)	(38)	(39)	(40)
(20) Y5 Psych Aggression	-																				
(21) Y5 Corp Punishment	.45	-																			
(22) Y5 Neglect	.29	.18	-																		
(23) Y5 IPV	.23	.19	.19	-																	
(24) Y5 ECV	.28	.22	.22	.14	-																
(25) Y5 Relationship Instability	.01	.07	.06	.19	.16	-															
(26) Y5 MD	.22	.13	.19	.33	.19	.18	-														
(27) Y5 Jail	.13	.19	.10	.10	.20	.01	.11	-													
(28) Y5 Poverty	.17	.14	.15	.12	.29	.08	.12	.33	-												
(29) Y5 Housing	.16	.10	.20	.35	.16	.15	.39	.18	.27	-											
(30) Y5 Food	.18	.07	.29	.24	.15	.05	.44	.17	.32	.55	-										
(31) Y9 Psych Aggression	.54	.34	.20	.20	.10	.02	.18	.07	.07	.14	.12	-									
(32) Y9 Corp Punishment	.35	.57	.16	.15	.25	.10	.18	.08	.08	.17	.10	.50	-								
(33) Y9 Neglect	.15	.16	.38	.20	.13	.03	.20	.08	.11	.22	.24	.41	.27	-							
(34) Y9 IPV	.04	.11	.16	.38	.02	.02	.23	-.06	.00	.09	.06	.23	.10	.14	-						
(35) Y9 ECV	.19	.12	.12	.12	.54	.06	.13	.18	.33	.18	.12	.18	.17	.11	.09	-					
(36) Y9 Relationship Instability	.06	-.02	.00	.07	.05	-.41	-.01	-.26	-.09	.03	-.09	.04	.00	.01	.20	.03	-				
(37) Y9 MD	.19	.16	.10	.17	.11	.02	.49	.09	.16	.22	.33	.24	.13	.19	.20	.17	.04	-			
(38) Y9 Jail	.15	.14	-.02	.09	.26	.02	.08	.74	.30	.18	.09	.08	.10	.07	-.07	.22	.14	.11	-		
(39) Y9 Poverty	.16	.16	.13	.10	.23	.06	.12	.27	.69	.22	.24	.07	.07	.09	.08	.35	.07	.23	.25	-	
(40) Y9 Housing	.15	.20	.06	.18	.20	.03	.23	.15	.21	.40	.34	.18	.16	.32	.20	.19	.03	.36	.16	.26	-
(41) Y9 Food	.10	.10	.22	.19	.11	.17	.34	.17	.26	.40	.60	.23	.22	.25	.23	.16	-.10	.48	.11	.35	.50

Table 2.10. List of Adversities in Each Domain

Domain	Infancy	Years, 3, 5 and 9
<i>Lack of Safety</i>	Unsafe neighborhood Intimate partner violence	Severe psych. aggression Severe corp. punishment Neglect Intimate partner violence Community violence
<i>Family Instability</i>	Single parent at birth Father incarceration Maternal depression	Parental relationship instability Father incarceration Maternal depression
<i>Economic Hardship</i>	Below poverty level Housing insecurity Food insecurity	Below poverty level Housing insecurity Food insecurity

REFERENCES

1. Reichman NE, Teitler JO, Garfinkel I, McLanahan SS. Fragile families: Sample and design. *Children and Youth Services Review* 2001;23:303–26.
2. Wechsler D. Wechsler Intelligence Scale for Children: WISC-IV ®. 4 ed. San Antonio, TX: Harcourt Assessment; 2003.
3. Dunn LM. Peabody Picture Vocabulary Test. 3rd ed. Circle Pines, MN: American Guidance Service; 1997.
4. Roid GH, Miller LJ. Leiter International Performance Scale-Revised. Wood Dale, IL: Stoelting Co; 1997.
5. Suglia SF, Duarte CS, Chambers EC, Boynton-Jarrett R. Cumulative Social Risk and Obesity in Early Childhood. *Pediatrics* 2012;129:e1173–9.
6. Straus MA, Hamby SL, Finkelhor D, Moore DW, Runyan D. Identification of child maltreatment with the Parent-Child Conflict Tactics Scales: Development and psychometric data for a national sample of American parents. *Child Abuse & Neglect* 1998;22:249–70.
7. Straus MA, Field CJ. Psychological aggression by American parents: National data on prevalence, chronicity, and severity. *Journal of Marriage and Family* 2003;65:795–808.
8. Straus MA, Stewart JH. Corporal punishment by American parents: National data on prevalence, chronicity, severity, and duration, in relation to child and family characteristics. *Clin Child Fam Psychol Rev* 1999;2:55–70.
9. Sweet J, Bumpass L, Call V. The Design and Content of the National Survey of Families and Households. University of Wisconsin--Madison. Center for Demography and Ecology; 1988. Retrieved 2015 Sept 2. Available from: <http://www.ssc.wisc.edu/cde/nsfhw/nsfh1.pdf>
10. Lloyd S. The Effects of Violence on Women's Employment. *Law and Policy* 1997;19:139-167.
11. Waldfogel J, Craigie TA, Brooks-Gunn J. Fragile families and child wellbeing. *The Future of Children* 2010;20:87.
12. Cavanagh SE, Huston AC. Family instability and children's early problem behavior. *Social Forces* 2006;85:551–81.
13. Craigie T-AL, Brooks-Gunn J, Waldfogel J. Family structure, family stability and outcomes of five-year-old children. *Families, Relationships and Societies* 2012;1:43–61.
14. Kessler RC, Andrews G, Mroczek D, Ustun B, Wittchen HU. The World Health Organization Composite International Diagnostic Interview Short-Form (CIDI-SF). *International journal of methods in psychiatric research* 2005;7:171–85.

15. Bureau UC. Survey on Income and Program Participation. Washington, DC: 1996.
16. Caldwell MB, Bradley HR. Home Observation for Measurement of the Environment: Administration Manual. Tempe, AZ: Family & Human Dynamics Research Institute, Arizona State University; 2003.
17. Leventhal T, Selner-O'Hagan MB, Brooks-Gunn J, Bingenheimer JB, Earls FJ. The Homelife Interview from the Project on Human Development in Chicago Neighborhoods: Assessment of parenting and home environment for 3-to 15-year-olds. *Parenting: Science and Practice* 2004;4:211–41.
18. Anderson PJ. Executive Functioning in School-Aged Children Who Were Born Very Preterm or With Extremely Low Birth Weight in the 1990s. *Pediatrics* 2004;114:50–7.
19. Aarnoudse-Moens CSH, Weisglas-Kuperus N, van Goudoever JB, Oosterlaan J. Meta-Analysis of Neurobehavioral Outcomes in Very Preterm and/or Very Low Birth Weight Children. *Pediatrics* 2009;124:717–28.
20. Brooks-Gunn J, Gross RT, Kraemer HC, Spiker D, Shapiro S. Enhancing the cognitive outcomes of low birth weight, premature infants: for whom is the intervention most effective? *Pediatrics* 1992;89:1209–15.
21. Plomin R. Genetics and general cognitive ability. *Nature* 1999;402:C25–9.
22. Plomin R, Spinath FM. Genetics and general cognitive ability (g). *Trends in Cognitive Sciences* 2002;6:169–76.
23. Wechsler D. Wechsler Adult Intelligence Scale - revised (WAIS-R Manual). Harcourt Brace Jovanovich; 1981.
24. Schlomer GL, Bauman S, Card NA. Best practices for missing data management in counseling psychology. *Journal of Counseling Psychology* 2010;57:1–10.
25. Little TD, Jorgensen TD, Lang KM, Moore EWG. On the Joys of Missing Data. *Journal of Pediatric Psychology* 2014;39:151–62.
26. Enders CK. *Applied Missing Data Analysis*. New York: Guilford Publications; 2010.
27. Razza RA, Martin A, Brooks-Gunn J. The implications of early attentional regulation for school success among low-income children. *Journal of Applied Developmental Psychology* 2012;33:311–9.
28. Costello AB, Osborne JW. *Best Practices in Exploratory Factor Analysis: Four Recommendations for Getting the Most From Your Analysis*. Practical Assessment, Research and Evaluation 2005;10:1–9.

29. Bollen KA. Structural Equations with Latent Variables. Canada: John Wiley and Sons, Inc; 1989.
30. Little TD. Longitudinal Structural Equation Modeling. New York: The Guilford Press; 2013.
31. MacKinnon DP, Lockwood CM, Hoffman JM. A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods* 2002;7:1-35.
32. Baron RM, Kenny DA. The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of personality and social psychology* 1986;51:1173–82.
33. Muthén BO, Muthén LK. *Mplus User's Guide*. Seventh Edition. Los Angeles: 2012.
34. Fritz MS, MacKinnon DP. Required sample size to detect the mediated effect. *Psychological Science* 2007;18:233–9.
35. Jeon J. The Strengths and Limitations of the Statistical Modeling of Complex Social Phenomenon: Focusing on Sem, Path Analysis, or Multiple Regression Models. *International Journal of Social, Educational, Economic and Management Engineering* 2015;9:1–9.

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CHAPTER 3

MULTIPLE ADVERSITIES AND CHILD COGNITIVE DEVELOPMENT: A CONCEPTUAL FRAMEWORK

INTRODUCTION

Children's social environments shape their cognitive development, including their general cognitive ability and executive functions (1,2). General cognitive ability (i.e., intellectual capacity, IQ) refers to general reasoning and thinking ability and is associated with one's ability to plan and solve problems (2,3). Executive functions are distinct neurocognitive processes including sustained attention, working memory and impulse control (4,5). These processes are integral to the development of behavioral self-regulation and social and cognitive competence (4). Given the importance of cognitive development for future success and its malleability in response to environmental input (1,2), there is increasing interest in understanding the mechanisms by which social context and experience influence cognitive outcomes.

Stress or adversity in early life can impair child cognitive performance (6,7). The negative influence of poverty on cognitive outcomes is among the most robust findings in developmental research (2,8). However, other adverse experiences, including abuse, neglect, family instability, parental mental illness, parental substance abuse, parental incarceration, domestic violence and neighborhood violence, also influence cognitive outcomes (9-11). Data from the National Survey of Children's Health show that nearly half of children ages 0-17 have experienced at least one such adversity, and nearly a quarter have experienced two or more (12). These adversities span social and economic classes, though many are common to conditions of poverty and may explain, in part, the relationship between poverty and poor cognitive performance (13).

Children exposed to *multiple* adverse experiences have worse cognitive outcomes relative to children with any single adverse exposure (14,15). The well-publicized

Adverse Childhood Experiences (ACE) Study showed that the number of adverse childhood experiences to which a child is exposed is associated with health risk behaviors and diseases in adulthood (16). Since the ACE study, evidence has accumulated to support the somewhat intuitive notion that as adversity exposures increase in a young person's life, negative health and developmental outcomes, including cognitive outcomes and achievement, are more likely to occur (11).

These findings have spurred increased interest among pediatricians and public health practitioners to screen children for early adverse experiences and to connect identified children to appropriate services (12,17,18). Such intervention efforts could be improved with a stronger conceptual model describing the relationship between multiple adverse exposures and child development. For example, across studies of adverse childhood exposures, the number and types of adversities studied often differ, as do the methodological approaches for examining the relationship between multiple adversities and cognitive outcomes. These differences present challenges for determining those adversities most important for screening and thresholds for referral, as well as their underlying mechanisms of action. Mechanisms are critical for informing and focusing intervention efforts.

Timing of adverse exposures may also matter. The brain develops rapidly in childhood, and research from both animal and human models suggests that when an area of the brain is rapidly developing, that region is more sensitive to environmental threats (6,7,19-21). Most studies examining the effects of multiple adverse experiences and developmental outcomes have disregarded the effect of timing of exposure (11,22). Studies are often cross-sectional or assess general exposures to adversity over a broad

developmental period (for example, any exposure prior to the age of 18 years). Thus, there is little empirical evidence about the existence of sensitive periods when children may be particularly vulnerable to multiple adverse exposures (11,22). This is essential for timing prevention programs when they will be most effective.

Aims of Current Review

The current review aims to answer three key questions: First, what are salient adversities to assess in the context of multiple adverse exposures? Second, what is known about underlying mechanisms or mediating pathways between multiple adverse exposures and cognitive outcomes? Third, what is known about the timing of multiple adverse exposures in relation to cognitive outcomes? The review summarizes general findings from the literature for each of these three questions and then proposes a conceptual model to guide future research and interventions in this field.

METHODS

The study of childhood adversity spans the fields of education, psychology, sociology, medicine and public health. Therefore, the PubMed, PsycINFO, CINAHL, Web of Science, and Scopus databases which span these fields were searched for variations of the following search strings: [“cumulative adversity” OR “cumulative risk” OR “adverse experience(s)” OR “adverse events” OR “stressful life events” OR “early life stress” OR “multiple risk” OR “adversity” OR “number of risk factors” OR “adverse childhood experience”] AND [“cognitive function” OR “cognition” OR “academic achievement” OR “attention” OR “school readiness” OR “memory” OR “learning” OR

“IQ” OR “Intelligence” OR “executive function” OR “inhibitory control” OR “cognitive control” OR “fluid intelligence” OR “fluid cognition”] **AND** [“children” OR “youth(s)” OR “child” OR “adolescent(s)”]. Titles and abstracts of the articles identified were screened for their relevance to the review topic. Relevant studies identified in review articles or in reference lists were also collected and screened for relevancy.

To be included in the review, articles met the following criteria: 1) published in English, in a peer-reviewed journal; 2) published from January 1990 to December 2013; 3) was a primary empirical report using quantitative methods; and 4) evaluated the multivariate relationship between three or more adverse experiences and at least one cognitive outcome, using a standardized assessment tool in children 18 years of age or younger. Adversities were defined as *exposures that typically create excessive demands or threats to the child but are preventable or amenable to change*, thus lending them to intervention. The requirement of a standardized assessment of cognitive outcomes was operationalized as having at least one standardized assessment tool with demonstrated validity and reliability of general cognitive ability (such as intellectual functioning or IQ) or specific executive functions (including measures of attention, impulsivity, inhibitory control, executive control, and working memory). Articles that described adversities in the context of war, natural disasters, or developing countries and non-human studies were excluded. Studies of timing were further limited to those that utilized a longitudinal design with adversities and/or cognitive outcomes assessed at multiple points in time. All studies that met these criteria were reviewed and scored for methodological rigor.¹²

¹² Scoring criteria were adapted from the Quality Assessment Tool for Quantitative Studies available at: http://www.ehph.ca/PDF/Quality%20Assessment%20Tool_2010_2.pdf. Studies that scores at least 10 out of 21 total points were included in the review (described in more detail in Chapter 2).

RESULTS

Summary of Studies

The combined search results yielded 3999 articles (not excluding duplicates). After screening the titles and abstracts for relevancy, 413 articles were reviewed in further detail to determine if the inclusion and exclusion criteria were met. Of these, 23 articles met the inclusion criteria. Table 3.1 summarizes the study designs of articles included in the review, the majority of which were longitudinal. Seventeen studies focused on general cognitive ability as the primary outcome, and seven studies looked specifically at executive functions (one study looked at both sets of cognitive outcomes).

Table 3.1. Number of Studies for Each Cognitive Outcome by Study Design

Cognitive Outcomes	Case-Control	Cross-Sectional	Longitudinal
General Cognitive Ability	1	5	11
Executive Functions	0	2	5

The studies included in this review assessed between three and 13 adversities. These included maltreatment (abuse or neglect), aspects of family structure and functioning (relationship instability, parental mental health, parental incarceration, parental substance use, housing mobility), poverty-related exposures (crowded dwelling, housing quality and instability, food insufficiency, low income-to-needs), school characteristics (unsafe climate, getting bullied), and neighborhood characteristics (violence exposure). While some studies examined factors across each of these domains, others focused more narrowly. Many studies also examined a number of risk factors (i.e., low birth weight, birth complications, teen or single parent, maternal cognitive ability or maternal education), and child characteristics (i.e., temperament, behavior problems). This review attempts to disentangle adversities from these other risk factors or child characteristics to strengthen our conceptual understanding of how adverse experiences

shape cognitive development. Tables A3.1 and A3.2 in the Appendix at the end of this chapter summarize the studies included in the review for outcomes of general cognitive ability and executive function, respectively. These tables also include the various adversity measures used in each study.

Studies examining the influence of multiple adverse experiences on child cognitive development have generally approached this question in three different ways, and each of these different approaches provides a different perspective on the influence of multiple adverse experiences on cognitive outcomes (see Evans, Li and Whipple, 2013, for an in-depth discussion of these methods). Most studies (including the ACE study cited above) use a cumulative index, constructed by dichotomizing each adverse exposure (1 = “exposed” and 0 = “not exposed”) and then summing the number of exposures into a single aggregate measure. The strength of the cumulative index is in its simplicity – one summary score that is easily understood and communicated to laypersons and policy makers. It is also useful for communicating information about the dosage of exposures. Multiple regression (or ordinary least squares) is a second methodological approach to studying multiple adversities. This method provides more information about the contribution of individual exposures than does the cumulative index approach but is less informative about the dosage of adversities. A third, domain-based approach, has also been used, though less frequently. A domain-based approach groups adversities of a similar type either by a cumulative index score (i.e., food insecurity, parental employment and poverty level combined to create one cumulative score for financial need) or other factor analytics. Domains provide information about dosage of effects as well as insight into the relative salience of particular types of adversities or the

relationship between these domains and mediating factors (11). Table 3.2 shows the number of studies that utilized each of these approaches for both sets of cognitive outcomes. A summary of the findings from these various approaches is provided below.

Table 3.2. Number of Studies for Each Cognitive Outcome by Methodology

Cognitive Outcomes	Multiple Regression	Cumulative Index	Domains
General Cognitive Ability	7	14	5
Executive Functions	3	4	3

Salient Adverse Experiences for Cognitive Development

General Cognitive Ability. Among 15 studies that explored the relationship between a cumulative index and general cognitive ability, 12 studies supported the general consensus that cognitive scores worsen as the number of adverse exposures increases (10,23-32). This gradient for the relationship between the number of adversities and cognitive ability was observed in studies from infancy through adolescence. Only one study showed no significant effect of a cumulative index at predicting concurrent cognitive ability (by Bayley Scales of Infant Development) among 12-month olds (33); one showed that the number of adversities was only associated with cognitive impairments among children and adolescents with ADHD (but not normal controls) (34), and another showed only marginal statistical effects of a cumulative index on concurrent cognitive functioning among ten year olds (35).

Exploring the contribution of separate adverse exposures, seven studies used multiple regression (or ordinary least squares) models to assess the relationship between independent adverse exposures and general cognitive ability (24,26,30,31,33,36,37). In these models, each adversity was entered into a regression model while adjusting for all other adversities, and the significance of each individual adversity, as well as overall

variance (or R^2) explained by the model, was used to evaluate effects. Multiple adverse exposures explained more of the variance in cognitive outcomes than any single adversity (33,38) and when compared to a cumulative index approach (26,30,33). Several adversities emerged as significant predictors of child cognitive ability, after accounting for other exposures, including those associated with low socioeconomic status (24,30,31), neighborhood safety (30), maternal depression (31,37), and characteristics of the family and home environment (such as child care quality and parenting behaviors) (24,26,30,37). However, such multiple regression-based studies are challenging to interpret because individual variables that may be otherwise significant will no longer appear so when other, correlated variables are included in the model. Results can overlook important but less influential adversities, leading to seemingly contradictory conclusions (26).

Five studies used a domain approach to examine the influence of multiple adversities on general cognitive ability. Only one study examined two theoretically determined domains of adversities – one human capital (maternal employment, education and welfare status) and the other psychological adversities (low social support, maternal depression, and stressful life events). While higher scores in both domains at birth independently predicted poorer cognitive scores at ages three, five and eight years, only the human capital domain predicted cognitive scores at all three ages after including both domains in the model. The human capital domain showed similar effects to a single cumulative index (comprised of all human capital, psychological and demographic factors), suggesting that a domain approach might provide more insight than a total cumulative index model (29).

Two other domain-based studies compared theoretically determined domains of psychosocial adversities to domains of other *risk factors*. For example, one study compared a biomedical risk domain (including perinatal and birth complications) to a psychosocial domain (including combinations of economic and maternal and family characteristics in early life), with each domain as a separate cumulative index. Children with higher scores in each of these domains had lower IQ scores at four years, and those who scored high in both domains had the worst effects (39). Another study compared a psychosocial domain (consisting of economic and maternal characteristics) against early neurocognitive measures (consisting of measures of a child's language, motor and memory skills) in early childhood. In this study, only the neurocognitive index significantly predicted WISC digit span scores at age nine (25).

Two additional studies used factor analysis to generate adversity domains (or factors) from the analytic sample. These empirically driven domains represent correlations among adversities in a particular sample and, therefore, may not generalizable to other populations. For example, In a case control study of children with and without ADHD, one study reported on two psychosocial factors; one factor consisting of maternal psychopathology, criminality and low SES predicted lower IQ scores among 6-17 year olds whereas a second factor consisting of family size and conflict did not (34). In a study of children under five years, three psychosocial factors were generated, including family risk (low maternal education, poverty, single status, and low maternal responsiveness to child); household size and maternal depression; and parenting stress and quality of child care. All three factors negatively predicted cognitive

development in a cross-sectional analyses across the first four years, but only the latter two domains predicted lower cognitive scores over time (26).

Executive Function. Four studies looked at the influence of a cumulative adversity index on some aspect of executive functioning. Three of these were longitudinal studies of children under five years using a cumulative index comprised of demographic, psychological and social risks and adversities. In the first of these studies, a cumulative index in the first and third years of life predicted worse attentional outcomes at age three (40). Another study showed that a cumulative index at age three predicted relatively smaller developmental improvements in effortful control over a six month period (41), and a third study found a cumulative index comprised of exposures over the first four and a half years predicted more impulsivity and worse attention at four and a half years (42). An additional cross-sectional study found that a cumulative index measure comprised of family psychosocial and physical adversities (noise, crowding, and substandard housing) predicted worse performance on a delay of gratification task among 8-10 year olds (43).

Three studies examined the influence of multiple adversities on some aspect of executive function using a multiple regression approach. These same studies also compared different grouping of adversities in relation to the outcome of interest. Therefore, they were also classified as domain-based studies. Two of these studies were longitudinal in design and interested in distinguishing the effects of poverty from other adversities on a child's executive functioning. One study found that poverty and a child's verbal ability (known to be associated with poverty) did not explain the variance in

inhibitory control among a low-income sample of children, whereas measures of family dynamics, including chaos and instability, did explain variance in inhibitory control (38). The other found that family poverty negatively influenced concurrent executive control from early to middle childhood; although there was no direct effect of school-related adversities (including safety and adult support), unsafe school environments moderated the effect of poverty such that unsafe environments exacerbated the effect of poverty on executive control (44). Worth noting, however, this study did not include any other measures of family adversities. A cross-sectional study of 10-12 year olds assessed eight different adverse experiences within their last year, including five personal stressors (emotional and physical abuse, emotional and physical neglect, parent and school stress), and two community stressors (neighborhood violence and other neighborhood problems) on a range of executive function measures. When looking at each cluster of stressors separately, both personal and community stressors were associated with behavioral measures of regulation (though neither cluster was associated with executive function); however, once both clusters were added to the model, only the personal stressors continued to have a significant effect (36).

Underlying Mechanisms or Mediating Pathways

General Cognitive Ability. Five studies examined mediating factors by which multiple adversities influence cognitive ability. Characteristics of the home environment and parenting emerged as important mediators of the relationship between exposure to adverse experiences and cognitive ability, and this was primarily true for poverty-related adversities. In a study exploring the multivariate effects of poverty-related adversities,

families that received government assistance, had unemployed parents or in which mothers had less than a high school education, were more likely to provide less cognitive and language stimulation in the home, leading to lower cognitive performance among children under three (24). In a path analysis testing the influence of distal and proximal adversities on cognitive competence in five year olds, the relationship between economic hardship and cognitive competence was mediated by the quality of the home environment (i.e., the extent to which the home environment was stimulating, safe and responsive). The home environment also served as a moderating factor in this study, such that children with high levels of risk but better quality home environments demonstrated better cognitive competence than children with high risk in low quality home environments (30). Additionally, the quality of the home environment mediated the relationship between the cumulative socioeconomic risks of caregivers and children's cognitive abilities among children (age 2-7 years) of incarcerated parents (32).

Specific parenting behaviors were also identified as mediators of the relationship between adversity and cognitive ability. Among low-income, rural infants, aspects of parenting, including maternal warmth, learning and literacy activities and maternal language (specifically, the diversity in vocabulary when interacting with the child) were significant mediators in the relationship between social adversities (including family demographics, stressful life events and neighborhood safety) at six months of age and the Bayley Scales of Infant Development score at 15 months (learning and literacy activities most consistently mediated this relationship at both 6 and 15 months of age). Specifically, families with more adversities provided less warm parenting, were harsher when interacting with the infant and used less diverse vocabulary. Adversity exposure more

strongly predicted less engaged parenting and fewer learning and literacy activities among a rural African American population relative to a rural Appalachian population suggesting potential differences in race and geographic location. In this study, positive parenting behaviors also buffered the effect of high social adversity on cognitive outcomes (27).

Another study explored multiple parenting behaviors under conditions of socioeconomic disadvantage, including poverty, stressful life events and maternal depression, and found that different adversities resulted in different parenting behaviors which, in turn, had unique effects on child cognitive outcomes. For example, non-responsive parenting mediated the effects of maternal depression on the cognitive outcomes of two year olds while there was no effect of intrusive or negative parenting. However, children of parents who were non-responsive but also more intrusive fared better than children of parents with low responsiveness and low intrusiveness. Additionally, children of parents with low negative parenting and high intrusiveness also did better than children with low negative parenting and low intrusiveness. These results suggests that nuanced patterns of parenting may result from socioeconomic disadvantage, with combinations of poor parenting behaviors having unique effects on cognitive outcomes (37).

Executive Function. Two mediation studies explored the role of parenting in explaining the relationship between a cumulative adversity index and child executive functioning. Using structural equation modeling, one study showed that both maternal warmth and the cognitive stimulation in the home partially mediated the effect of early

adversity in the first year of life on a latent construct of attention and behavioral regulation at age three (40). Another study focused on four dimensions of parenting and found that limit setting and scaffolding mediated the relationship between the cumulative adversity index and executive control among preschoolers, but maternal warmth and negative affect did not (41).

Timing of Adverse Exposures

Overall, twelve longitudinal studies explored some aspect of timing when examining the relationship between multiple adverse exposures and cognitive outcomes. All of these studies assessed children under five years of age; three studies extended follow-up into middle childhood (six to eight years), and one study followed children to age thirteen years.

General Cognitive Ability. Among the eight studies of general cognitive ability, five explored the relationship between adverse exposures at a single point in time in relationship to concurrent and subsequent cognitive abilities (28-30,37,39). Overall, concurrent adverse exposures were most detrimental to general cognitive ability, and the predictive power diminished over time. However, one study showed that for adversities related to lower human capital, the opposite may be true; lower human capital at birth had the most pronounced effect on cognitive ability at later time points, most notably when a child entered school (29). Among studies that measured both adversities and cognitive ability at multiple points in time, both adversities and cognitive ability were moderately stable (10,26). One study showed disparities in cognitive scores between children with

high and low levels of adversities increased as children aged, with impairments in cognitive performance evident as early as 14 months (24).

Executive Function. Four studies examined the relationship between multiple adversities and executive functions over time (40,41,44,45). These suggest that executive functions are malleable and generally improve with age for all children in early and middle childhood; however, children with more adverse experiences show the least improvement in executive functions over time (38,41,44). Additionally, children with detriments to executive function early in life due to adversities in the family may be more susceptible to school-related adversities that arise in middle school (44). Only one study explicitly explored the influence of sensitive periods (40). It found that a cumulative index (comprised of economic factors and maternal mental health) at twelve months explained more of the variance in a composite measure of attention at three years than did the cumulative index at age three, providing evidence for the significance of early adversity exposure.

DISCUSSION

An array of terminology is used to capture the phenomenon of children with multiple adverse exposures, including risks, adversities or stressful life events. Each of these terms potentially encompasses different categories of exposures, presenting a challenge for drawing comparisons across studies. Additionally, it is important to distinguish adverse exposures from the underlying mechanisms or processes that may explain *how* adversities influence developmental outcomes. For example, among the

studies reviewed, several referred to aspects of parenting, such as the level of parental warmth or cognitive stimulation in the home, as measures of adversity while others referred to these characteristics as mediators of the relationship between other adverse exposures and child outcomes. Recognizing that it is difficult to disentangle these nuances, the conceptual model makes a distinction between adverse exposures and resulting parenting behaviors that are better classified as mediating factors.

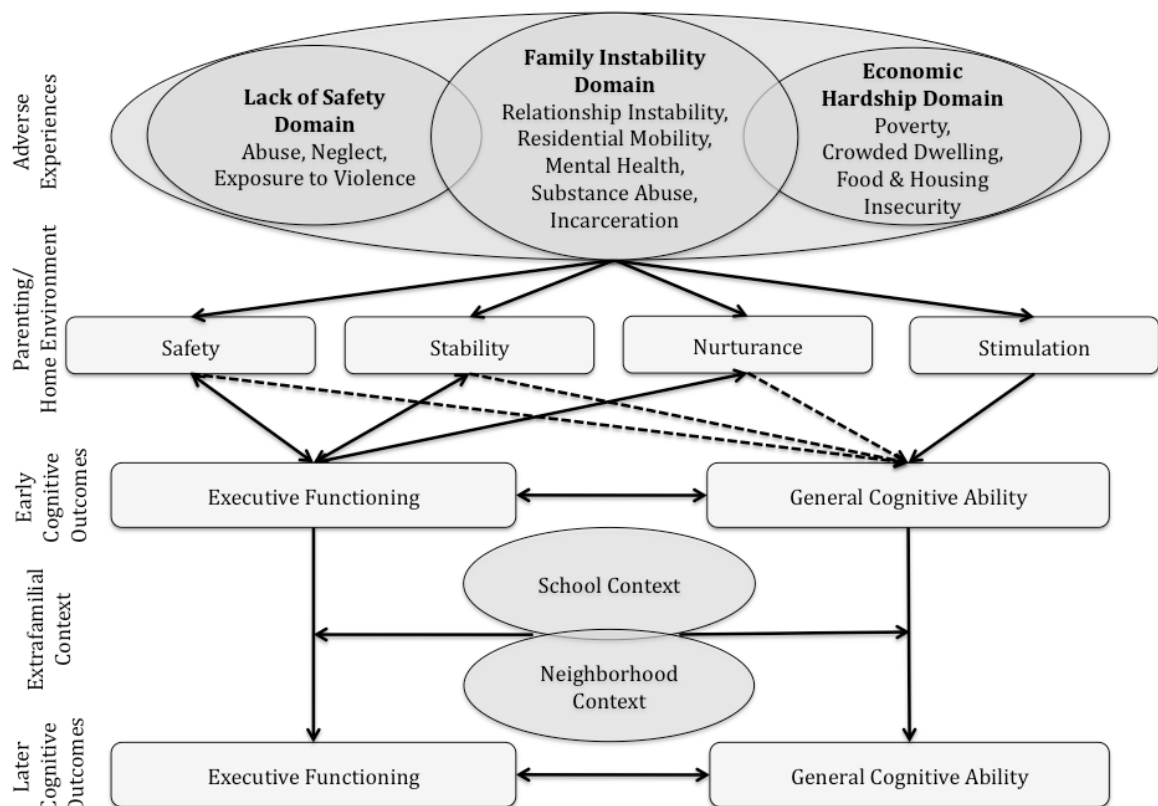
A Conceptual Model

Figure 3.1 synthesizes the findings from this review into a proposed conceptual model to guide future research and interventions on multiple adversities and cognitive development.

Adversity Domains. A domain-based approach to studying multiple adverse exposures is promising (11). While few studies have taken this approach, the handful in this review showed unique effects of individual domains on cognitive outcomes, though there was insufficient consistency across studies to draw significant conclusions about how adversities should be grouped together or the particular salience of particular domains. A recent study, however, examined the factor structure of the 11-item Adverse Childhood Experiences (ACE) module of the Behavioral Risk Factor Surveillance System from a sample of nearly 30,000 adults. The study found evidence for a three-factor structure, grouping adversities into a household dysfunction domain (consisting of household mental illness, alcohol and substance abuse, incarceration, and parental separation/divorce), a physical and emotional abuse domain, and separate domain for

sexual abuse (46). This content-specific grouping provides a useful framework upon which to conceptualize adversity domains. However, the ACE module may also be incomplete in that numerous adversities that may be relevant to child cognitive development are not assessed, including poverty level and exposure to community violence.

Figure 3.1. Conceptual Framework



Building upon this framework and the findings from this review, the proposed conceptual framework describes three domains of adverse exposures. The first pertains to economic hardship (such as living at or below the poverty threshold, living in a crowded dwelling, and having food or housing insecurity). Nearly all of the studies included in this review directly measured some aspect of economic resources as an adverse exposure. One could argue that poverty is not an adversity, but rather a distal

factor that broadly describes a context in which other adversities are more likely to occur. However, among children who exhibit the same levels of adverse exposures, but differ only in their level of poverty, those living in poverty tend to have worse cognitive outcomes suggesting that severity of poverty compounds the detrimental effect on cognitive development (10). Therefore, economic hardship is included in this model as a unique domain. The second domain pertains to family instability. This domain includes adversities such as parental relationship instability, residential mobility, parental mental health or substance abuse disorders, or parental incarceration. As demonstrated in this review, these factors were critically related to cognitive outcomes, and were organized into a single domain in the ACE study described above. The third domain includes exposures that directly threaten a child's physical safety, including the exposure to abuse, neglect, domestic violence or community violence. While few studies included in this review measured aspects of this third domain, there is sufficient research elsewhere to suggest that the chronic stress associated with exposure to violence or maltreatment has unique detrimental effects on child cognitive development (7,47,48).¹³ These particular domains may have more salience in early childhood, when the child's social context consists primarily of the family. As a child's social world expands, additional adversities outside of the family context may be worth exploring, including school-related adversities (such as bullying or school violence) or neighborhood-level adversities (such as a crime). School and neighborhood context may also serve as protective factors or supports (44).

¹³ Studies that met the criteria for this review tended to focus more heavily on adversities related to socioeconomic disadvantage or family structure and function. In hindsight, the search terms for this review could have been expanded to include "polyvictimization" in order to capture multiple types of exposures to violence. However, the lack of studies that included adversities across all three domains was surprising.

While not depicted in this model, other factors, not classified as adversities (such as maternal education or parental cognitive ability), are also strongly associated with child cognitive development and should be accounted for in any study of adverse exposures and child cognitive performance (49).

Malleable mechanisms. Among the studies included in this review, the quality of the home environment and specific parenting behaviors emerged as important mediators of the relationship between multiple adverse experiences and cognitive performance. The proposed conceptual model draws upon these findings as well as other existing frameworks to suggest that the adversity domains described above influence developmental outcomes through parenting and the home environment in ways that disrupt the safety, stability, nurturance and stimulation provided to a child. The Centers for Disease Control and Prevention developed a framework for the prevention of child maltreatment that emphasized the importance of safe, stable and nurturing relationships for healthy development (50). *Safety* refers to the extent to which a child is free from fear and secure from physical or psychological harm within their environment. *Stability* refers to the degree of predictability and consistency in a child's environment. *Nurturing* refers to the extent to which a parent or caregiver is available and able to sensitively and consistently respond to and meet the needs of their child. While not a part of the CDC's model, *stimulation* is another important mechanism by which adversity can influence child cognitive development. Stimulation refers to the level of learning experiences available in a child's environment.

One of the more consistent findings from this review was the mediating pathway from poverty-related adversities to cognitive ability through cognitive stimulation in the home. A body of research on the effects of poverty on child cognitive development supports this finding. Poverty may limit the capacity of families to invest in stimulating home environments (i.e., books, activities) as well as other resources and services that benefit child health, including nutrition, housing, health care, and child care (8,51,52). In addition, family stress may interfere with parents' ability to allocate time and energy to interact positively with their children, thus influencing the safety, stability and nurturance provided (52,53). Similarly, unstable home environments, which are often closely linked with poverty, may also impair cognitive development by disrupting positive parent/child interactions (54).

The relations between lack of safe, stable and nurturing environments and child cognitive development may be partly due to alterations in the stress response. In response to threat, sensory information from the environment is translated into a set of cognitive, behavioral, and physiological responses that are critical to survival; however, continuous engagement of the stress response may inhibit cognitive, behavioral, and physiological adaptation in the long term (6,55,56). One of these responses, the hypothalamic-pituitary-adrenal (HPA) stress response is well-studied (6). Cortisol, the end-product of the HPA cascade, acts throughout the body and brain (6,47,56). Cortisol receptors are densely expressed in the hippocampus, prefrontal cortex, and amygdala – regions of the brain that regulate the HPA axis (6).

Studies have documented atypical stress reactivity among children exposed to multiple adversities (57,58), unsafe environments caused by violence and maltreatment

(7,59,60), and poverty (21). Animal models suggest that overproduction of cortisol in response to chronic stress and the underproduction of cortisol that may arise from severe deprivation can inhibit neurogenesis in the hippocampus and the prefrontal cortex, negatively impacting learning, memory, and cognition (6,7,55,56). Parent-child interactions can also influence HPA activity through child attachment. More responsive parenting has been shown to yield more securely attached children, and more securely attached children are less reactive to acute stressors (55,57,61).

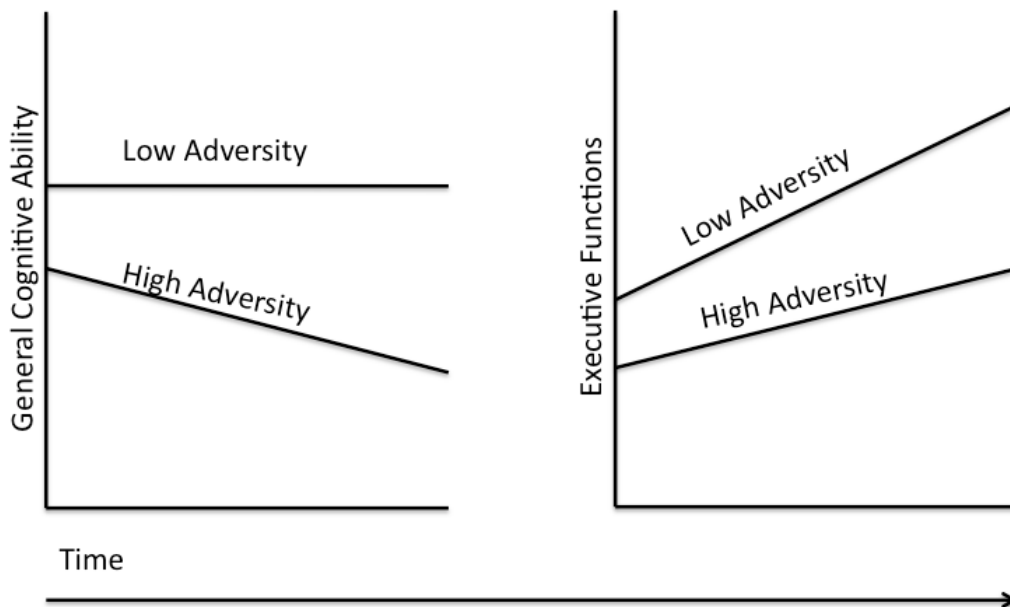
Other mechanisms not depicted in this model include prenatal exposures (such as substance abuse, low birth weight, and early gestational age) (62,63), chemical exposures (such as lead exposure) (64), and malnutrition or poor nutrition (8). These factors are known to be associated with both poverty and cognitive development (8). However, less is known about whether these factors explain the relationship between other adversity domains and child cognitive development.

Timing of Adverse Experiences. Few studies in this review explored the temporal nature of adverse exposures on cognitive outcomes, and even fewer measured both adverse exposures and cognitive outcomes at multiple points in time. Although not conclusive, several interesting findings emerged. First, longitudinal studies that measured cumulative adversities at multiple points in time showed the total number of adverse exposures to which a child is exposed to be moderately to highly stable over time (e.g., the total number of adversities at one time point was similar to the total number of adversities at another time point) (10,26,40). However, the exposures accounted for in these studies were primarily related to socioeconomic status (i.e. maternal education, race,

or poverty level). Future studies would benefit by examining the stability and temporal influence of adverse experiences further, particularly the stability of different adversity domains.

Second, different developmental trajectories were observed for both cognitive ability and executive function among children with high and low levels of adversity. These trajectories are illustrated in Figure 3.2. In general, cognitive ability was moderately to highly stable over time, with more stable performance for children with less adversity. Although changes in cognitive ability over time may reflect malleability in response to experience, developmentally appropriate assessments of cognitive performance may also introduce measurement variability across distinct developmental periods. The effects of adverse experiences had the greatest influence on assessments of general cognitive ability at the time in which the exposures occurred. Over time, however, the effect of early adverse exposures diminished. When there was documentation that adversities continued to persist over time, deficits in cognitive performance among children with high exposure increased. Unlike general cognitive ability, executive functions continued to improve over time for all youth, which is consistent with the development of the prefrontal cortex throughout childhood and adolescence. However, exposure to early adversity impeded the rate of maturation of executive functions. There was not enough research to determine whether these deficits in cognitive ability or executive functioning were recoverable.

Figure 3.2. Influence of Multiple Adverse Experiences and Cognitive Development Over Time



Developmental research offers differing perspectives with respect to timing of multiple adverse exposures in relation to shaping outcomes later in life (65). The *revisionist perspective* maintains that early experiences are important in the short-term or at the time that the adversities are experienced, but as children mature, they acquire new competencies and are faced with new experiences that weaken the association of early exposures with later outcomes (65). For example, excessive stress narrows attention on the source of threat and limits the bandwidth for other cognitive functions – at the time the stress is experienced (66). On the other hand, the *enduring effects perspective* suggests that early experiences are preserved over time, leading to long-term associations of early exposures with later outcomes (65). Enduring effects may be explained by alterations in neurobiological regulatory systems during times of plasticity or by “allostatic load”, where chronic or repeated exposure to psychosocial stressors over time

leads to wear and tear on the body that results in dysregulation of various regulatory systems, including cognitive functioning (67).

The findings from this review provide some support for both the revisionist and enduring effects perspectives. Among those studies that examined the relationship between adversities at a single point in time and general cognitive ability over time, there was some evidence to suggest that the effects of early adversities diminished, in line with the revisionist perspective. However, among studies that examined both adversities and general cognitive ability over time, there was more support for the enduring effects perspective—that is, exposure to earlier adversities was associated with diminished cognitive ability or less maturation in executive functions over time. This difference may be attributed to the finding that adversities are likely to persist over time, leading to more continuous or chronic exposures. Only one study from this review explicitly evaluated sensitive periods for cumulative exposures and found evidence for a sensitive period in the development of attention and regulation in the first fourteen months of life (40). This adds to a growing body of evidence that adversity in early childhood has lasting effects on cognitive outcomes (68). However, this does not negate the possible harm of later adverse exposures. Further research is needed to tease apart the differential effects of multiple adversities at a single point in time versus chronic exposure over extended periods of time.

Implications and Conclusion

The conceptual framework presented here has implications for both research and practice. From a research perspective, the framework proposes relevant domains of

adversities for studying cognitive development. Further examination of these adversity domains will help identify intervention targets. As pediatricians and public health providers embark on increased efforts to screen children for adverse experiences and link them to appropriate care, interventions may be more or less effective, depending upon the constellation of adversities to which a young person is exposed. Rather than directing a child to services based on the child's overall adversity score, attention must be given to the *types* of adversities experienced.

The model also distinguishes adversity domains from other underlying mechanisms. The articles included in this review focused primarily on the mediating role of parenting behaviors in the relation between adverse experiences and cognitive development. Future research could add to this field of study by examining other potential mediators, such as nutritional quality. Additionally, future studies could examine gender differences in the relations between adversities and cognitive development as well as differences in underlying mechanisms.

While not a focal area of this review, more research is also needed on factors that protect against or buffer the effect of adverse experiences. Interventions that promote positive parenting practices in the home environment and enhance cognitive stimulation have been successful. Home visiting programs, for example, are designed to intervene with high-risk families early in a child's life, and have improved parenting practices that shape future outcomes for children (69). Additionally, early childhood education programs that aim to provide children with early experiences and stimulation are associated with better cognitive outcomes among children who have experienced adversity (24,29) and have reduced disparities in achievement evident by the time that

socioeconomically disadvantaged children enter kindergarten (70). As children age, interventions that promote safe school and neighborhood environments may also buffer the effects of adverse exposures (44).

This review also underscores the need for more research on the timing of adverse exposures in relation to developmental outcomes. While neuroscience indicates that the timing of environmental input can significantly affect developmental pathways (6,7,20), we still know very little about this topic with respect to adverse exposures. The studies included in this review support the notion that both general cognitive ability and executive functioning are shaped by experiences over time, and adverse exposures as early as the first year of life, particularly related to poverty, alter developmental trajectories across childhood. However, more research is needed on the stability and temporal influence of adverse exposures in order to inform the timing of interventions efforts and make the most use of limited intervention resources.

APPENDIX, Table A3.1. Studies of Multiple Adversities and General Cognitive Ability

C=Comparison study; CI=Cumulative Index; D=Domain; I=Intervention; Int=International; M=Mediation; Mod=Moderation; MR=Multiple Regression; T=Timing

Article	Score/ Codes	Sample & Design	Adversity Measure(s)	Cognitive & Other Outcomes	Mediating & Moderating Factors, Covariates	Findings
Alaimo, Olson & Frongillo, 2001	15 CI, Mod	N=2063 children and teens from the Third National Health and Nutrition Examination Survey (NHANES III) cross- sectional	<p><u>6-11 Years:</u> <i>food insufficiency</i> <i>cumulative index (12 items):</i> unemployed family head, unmarried family head, crowded dwelling (>1 person per room), moved 3+ times in lifetime, no health insurance, no regular source of health care, blood lead exposure, low birth weight, exposure to prenatal smoke, birth complications, mother <18 years at birth, no child care attendance</p> <p><u>12-16 Years:</u> <i>food insufficiency</i> <i>cumulative index (8 items):</i> unemployed family head, unmarried family head, crowded dwelling (>1 person per room), moved 3+ times in lifetime, no health insurance, no regular source of health care, blood lead exposure, no child care attendance</p>	<p><u>6-11 Years and 12-16 Years:</u> <i>cognitive ability:</i> Wechsler Intelligence Scale for Children-Revised (WISC-R): block design, digit span <i>academic achievement:</i> Wide Range Achievement Test-Revised (WRAT-R) - reading and arithmetic <i>psychosocial outcomes:</i> number of days absent from school, visit for emotional, mental or behavioral problems, suspension from school, number of good friends, difficulty getting along, and somewhat shy to make friends</p>	<p>Moderator(s) CI x food insufficiency</p> <p>Covariates sex, age, race- ethnicity, health status, metropolitan region</p>	<p>Unadjusted relationship between food insecurity and all outcomes showed that food insufficiency was associated with worse WRAT and WISC scores as well as psychosocial difficulties for both children and teenagers. After adjusting for all other items in the cumulative index, food insecurity was associated with lower WRAT arithmetic scores, and having repeated a grade and seen a psychologist for children and several psychosocial problems for teenagers. There were no effects on cognitive or academic outcomes for teenagers. Effects were not accounted for by diminished health status.</p> <p>With increasing risk factors, both children and teens scored lower in WISC and WRAT. Only younger children also had more psychosocial problems with increasing risk.</p> <p>Food insecurity was most strongly associated with poorer academic outcomes among children with fewer other risks.</p> <p>Authors suggest that food insecurity is another risk factor that should be included in studies of multiple adversities.</p>
Aro, Poikkeus,	14	N=190	<p><u>4-6 Years</u> <i>parenting index (6 items):</i></p>	<u>8-9 Years</u>	Moderator(s) familial risk of	Total CI predicted all outcomes in expected direction. No differences in effects by gender

Eklund, Tolvanen, Laakso, Viholainen, Lyytinen, Nurmi, Ahonen & 2009	CI, D, Int	Finnish children (ages 1-9), half of whom had familial risk for dyslexia; primary language Finnish	maternal education, father unemployment, general stress, parenting-related stress, depression symptoms, parental sensitivity, support for joint attention, self reported affection in parenting	child's IQ: WISC digit span reading fluency (reading out loud) social adaptive behavior (parent report)	dyslexia, gender Covariates familial risk of dyslexia, gender	or familial risk. The neurocognitive domain predicted IQ and reading fluency whereas the parenting index predicted social adaptive behavior. The total CI did not have significant incremental effects on any of the outcomes above and beyond the effects of the individual domains. Authors conclude the effects were domain specific. Results do not support the model that each additional risk/adversity would increase generic stress.
		longitudinal	<u>2-6 years</u> <i>neurocognitive functioning index (6 items):</i> global language skills at 2 and 2.5 years, global language skills at 5 and 5.5 years (PPVT), phonological awareness at 3.5 years, phonological awareness at 5.5 years, rapid serial naming at 5.5 years, visual motor skills at 5 years, motor skills at 6.5 years, memory at 5 and 5.5 years (digit span) <i>total CI:</i> parenting and neurocognitive items combined			
Ayoub, O-Connor, Schlichtmann, Valloton, Raikes, Chazan-Cohen & 2009	18 MR, CI, M, T	N=2764 mothers and their children living in poverty - EHS Research and	<u>14, 24 and 36 months:</u> <i>microsystem index (4 items):</i> child negative emotionality (14 mo only), parent-child dyadic mutuality (synchronicity, comfort, and enjoyment; 14 and 24 mo only), cognitive and language stimulation in the home	<u>14, 24 and 36 months:</u> <i>Mental Development Index subscale of the Bayley Scale of Infant Development:</i> provides a score of the child's level of cognitive skill development relative to national norms	Mediator(s) earlier measures of adversity Moderator(s) EHS enrollment Covariates	Cognitive skills, on average decreased in the first 3 years of life in relation to national norms. At the exosystem level, SES-related risks predicted cognitive skills. Government assistance and low maternal education associated with cognitive skill decline from 14 to 36 mo and lower cognitive skills at 36 mo. Parental unemployment associated with lower cognitive skill at 36 mos. Effects of SES on 36 mo cognitive skills were mediated by

			Evaluation Project	(HOME scales)		child gender and ethnicity	stimulation at home and parent-child interactions. At the microsystem level, greater negative emotionality was associated with poor cognitive skills and a more rapid decline over time. Children enrolled in EHS performed higher on cognitive skill tests though effects were small. EHS did NOT moderate effects of risk – rather was a promotive factor no matter what the risk level. Stimulation in the home mediated effects of EHS. Using an additive CI measure, more risk related to worse cognitive skills. EHS did not buffer effects of CI on cognitive skills.
			longitudinal	<i>exosystem index (4 items):</i> EHS enrollment (time-invariant), reliance on government assistance, unemployment, low maternal education			
Biederman, Milberger, Faraone, Kiely, Guite, Mick, Ablon, Warburton & Reed, 1995	10	N=260	CI, D	<u>Ages 6-17:</u> <i>cumulative index (5 items):</i> severe marital discord, low social class, large family size (3 or more children), paternal criminality, maternal mental disorder Factor analysis yielded 2 factors: factor 1 (maternal psychopathology, paternal criminality, and SES) and factor 2 (family size and family conflict)	<u>Ages 6-17:</u> <i>full-scale IQ:</i> Wechsler vocabulary and block design <i>freedom from distractibility IQ:</i> Wechsler arithmetic, digit span and coding <i>psychopathology:</i> ADHD, conduct disorder, major depression, and anxiety <i>Other cog outcomes:</i> learning disability, repeated grades, in-school academic tutoring	Moderator(s) family history of ADHD Covariates family history of ADHD, age	For CI analysis, the odds ratio for ADHD, ADHD-related psychopathology, and psychosocial dysfunction increased with each increase in number of adversity indicators. There was a moderate increase in learning disabilities and cognitive impairment with increasing adversity. Factor analysis: Both factors predicted ADHD. Factor 1 associated with learning disability and cognitive impairment, while factor 2 was not. Impact on non-ADHD outcomes appears to occur independent of ADHD status. However, ADHD moderated effect of adversity on IQ; IQ decreased with increasing adversity among ADHD sample, but not controls.
Burchinal, Roberts, Hooper, Zeisel & 2000	15	N= 75	MR, CI, D, C, T	<u>1, 2, 3 and 4 Years:</u> <i>cumulative index (9 items):</i> father absent in household, large household, mother less than high school education, high maternal stress, high maternal depression, low	<u>1, 2, 3 and 4 Years:</u> <i>Cog development:</i> Bayley Scales (1 yr), revised Bayley Scales (2, 3 yrs), Weschler Verbal IQ (4 ys)	Mediator(s) Maternal IQ (Weschler), gestational age	Very high to moderate levels of across-time stability were observed in the CI, maternal education, household size, maternal depression, family environment quality, and child-care quality. Only stressful life events and maternal responsiveness during child interactions showed modest across time

care centers

longitudinal

maternal responsiveness in interaction with child, poverty, low levels of stimulation and responsiveness in the home environment (HOME), and low levels of stimulation and responsiveness in the child care environments.

*Risk stability over time so each risk factor averaged across time points

*Risk determined by distributional cut-offs

*Factor analysis also done for each age - 3 factors created: Family Risk, HH Size/Depression, Child Care/Stress

Expressive and Receptive Language: Sequenced Inventory of Communication Development (1,2, 3 yrs)

correlations. The child outcomes showed moderate stability across time. Three analytical methods compared: 1) individual variables, 2) factor scores derived from risk variables, and 3) cumulative index. All three analyses provide evidence that multiple adversities are more likely to lead to less optimal cognitive and language development and tend to score lower over time on language and cognitive measures. Individual variable approach provided better prediction at a particular age, but less useful in predicting developmental patterns.

Both factor and CI approaches better at predicting patterns of change over time, and CI better for a larger number of variables.

Burchinal, Vernon-Feagans, Cox & Key Family Life Project Investigators, 2008	17	N=1292	<u>6 months:</u> <i>cumulative index (7 items):</i> maternal education, family income, single parent, number of children in household, stressors or negative life events, parental unemployment, neighborhood safety *created both a count score and a composite score using mean of each variable	<u>15 months:</u> Bayley Scales of Infant Development	Mediator(s) 6 and 15 months: <i>5 parenting measures (assessments using HOME scale and observing parent-child interaction):</i> maternal engagement, harsh parenting, parental warmth, access to learning and literacy skills, maternal language	Cognitive skills were lower when children were exposed to more social adversities/risk, adjusting for other the confounders. Adding all four parenting skills to the model reduced the association between the CI and cognitive outcomes. Engaged parenting, increases in positive parenting and decreases in harsh parenting provided independent prediction (mediation). Learning and literacy and maternal language at 6 mo mediated relationship between the CI and cognitive outcomes. At 15 mo, parental warmth and learning and literacy mediated the association. Authors conclude that the severity of exposure to CI is related to poorer cognitive development for infants as young as 15 months, apparently through aspects of parenting (maternal warmth, learning and
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					Covariates ethnicity, region, geographic isolation, age	literacy activities, maternal language). Learning and literacy was the most consistent predictor (at 6 and 15 months). Mean measure of CI better predictor than count. Families with more exposures provided less warm and engaged parenting, were harsher when interacting with the infant and used less diverse vocabulary. No evidence emerged that parenting served as a protective factor (moderator) in contrast to some other studies.
Crozier & Barth, 2005	14	N=2368	<u>10 Years:</u> <i>cumulative index (5 items):</i> family poverty, prior involvement with welfare system, caregiver mental health problems, CBCL clinical behavior problems, teacher report clinical behavior problems	<u>10 Years:</u> <i>cognitive functioning:</i> Kaufman Brief Intelligence Test (KBIT) - verbal and nonverbal intelligence) <i>academic achievement:</i> Woodcock-McGrew-Werder Reading and Math	Mediator(s) KBIT a mediator of CI and academic achievement Covariates age, gender, maltreatment type	Outcomes for maltreated children were lower compared to the national average. No moderation by age or gender. Race was significantly associated with lower scores on the KBIT, but not reading or math tests. No effects of maltreatment types. With respect to individual risks/adversities, only poverty predicted lower scores on KBIT as well as math and reading. Prior child welfare and poor mental health predicted low reading and math scores. CBCL and teacher report of clinical problems predicted low math. CI was associated with low scores in reading and math, and marginal for KBIT after controlling for gender and age. Boys scored lower than girls. Impact of CI on reading and math partially mediated by KBIT.
Fishbein, Warner, Krebs, Trevvarthen, Flannery & Hammond, 2009	14	N=553	<u>Age 10-12:</u> <i>any abuse:</i> Childhood Trauma Questionnaire <i>any neglect:</i> Childhood Trauma Questionnaire <i>school stress (made up of 5- items):</i> trouble with teacher, getting suspended, being	<u>Age 10-12:</u> <i>Ravens Coloured Progressive Matrices:</i> nonverbal measure of general intelligence <i>Cambridge Decision-Making Task</i> (risk-taking)	Covariates child age and parent level of education	Bivariate relationships of stressors with cognitive outcomes showed higher levels of each stressor related to worse behavioral regulation. Abuse significantly related to lower Stop-Change impulsivity scores, and neglect significantly related to lower Raven's IQ and Ekman's facial recognition. In multi-variate regression (adjusting for all other stressors and covariates), only neglect was significantly

			cross-sectional	bullied <i>parent stress (made up of 7 items):</i> fighting, parent involvement, helping with homework, feeling that they cared <i>witnessing neighborhood violence</i> <i>perception of problems within neighborhood</i>	<i>Tower of London Test:</i> procedural memory and problem solving <i>Stroop Color Word Test:</i> flexibility and resistance to interference <i>Logan Stop-Change Task:</i> inhibition that involves ability to shift responses in light of new information <i>Ekman Facial Recognition Test:</i> accurately identify emotional expressions <i>Dysregulation Inventory:</i> emotional, behavioral, and cognitive dysregulation; only used impulsivity subscale in current study; measure of ADHD symptoms		associated misattributions of emotion during the facial recognition task, and physical abuse was related to lower problem solving scores. All stressors except neglect and neighborhood stressors were related to behavioral regulation. Authors conclude that stressor types are differentially associated with different neurocognitive tasks and future studies should take into account these much more nuanced and complex relationships.	
Hall, Sammons, Sylva, Mehuish, Taggart, Siraj-Blatchford & Smees, 2010	17	N=2899	CI, C, T	British children (36-58 months) longitudinal *comparison of CI with 2 latent composite indices (dichotomous vs. ordinal)	<u>36 months:</u> <i>cumulative index (11 items):</i> first language not English, 3 or more siblings, premature at birth, low birth weight, low mother education, father social status, father unemployment, teen mother, single parent, mother unemployment, bottom quartile home learning environment	<u>36 months:</u> <i>general cog abilities:</i> measured by British Ability Scales <u>58 months:</u> <i>general cog abilities:</i> measured by British Ability Scales	Covariates gender, ethnicity	Factor analytics comparable with the measures derived from cumulative indices. All three risk metrics predicted cog ability, but with some differences; the less assumptions made in the measure, the better the predictor. All three explained more of the variance in cog abilities at 36 months and less so at 58 months (but cognitive ability at 58 months controlled for the earlier assessment). Authors conclude that formative confirmatory factor analysis retains and returns more information than a cumulative index.

		risks)				
Hooper, Burchinal, Roberts, Zeisel & Neebe, 1998	10	N=83	12 months: <i>cumulative index (10 items):</i> income-to-needs (poverty status), maternal education (<12 years), household size (≥4 non-parents), single marital status, ≥20 stressful life events in past year, maternal depressed affect (90 th percentile), quality of mother-child interactions, maternal IQ (WISC), quality of home environment, quality of day care environment (HOME)	12 months: <i>cognitive development:</i> Bayley Scales of Infant Development <i>language development:</i> two standardized measures (SICD & CSBS); SICD provides overall measure of receptive and expressive language and CSBS provides communicative, social affective and symbolic skills of children	Covariates gestational age at birth	CI was associated with both language outcomes, but not the cognitive development measure. The multiple regression model significantly predicted the cognitive development outcome and the SICD language outcome (but individual risk factors not shown due to small sample size). Out of 10 factors, 9 were modestly related to the cognitive and language outcomes. Stressful life events was the only factor that did not relate to any outcome. The CI a better predictor of the CSBS outcome than the multiple risks model. However, the multiple risk model outperformed the CI model for the other two outcomes.
Klebanov & Brooks-Gunn, 2006	16	N=228	At birth: <i>human capital index (3 items):</i> maternal unemployment, welfare receipt, less than high school education <i>psychological index (3 items):</i> mental health (upper quartile), stressful life events (2 or more), low social support (lowest quartile) <i>demographic index (3 items):</i> teenage motherhood, father absence, # of children in household (4 or more)	3, 5 and 8 Years: Stanford-Binet Scale Form L-M for measure of cog performance (age 3) Wechsler Preschool and primary scale of intelligence (age 5) WISC (age 8)	Moderator(s) IHDP home visiting program and center-based care Covariates study site, sex, race, neonatal health, mother's immigrant status	Only welfare receipt and mothers having less than high school education significantly associated with cognitive test scores at ages 3, 5, 8 (controlling for covariates). Greater total CI was associated with lower test scores at 3, 5, and 8 years. When comparing the human capital and psychological indices, only human capital was associated with cognitive outcomes at ages 3, 5, 8 years. The psychosocial index was not significantly associated with cognitive outcomes at any age although slope was in the right direction. When both domains were included in the model, human capital accounted for more than 80% of the variance. The IHPD intervention was associated with cognitive outcomes at 3, 5, and 8, even after controlling for CI, but the intervention did not moderate the effects of CI. Treatment effects were found at 3 years (when the IHDP intervention ended), after

Krishnakumar & Black, 2002	14	N=217	low-income, AA mothers and their children recruited from two inner-city pediatric clinics	<u>5 Years:</u> <i>cumulative index (6 items):</i> family economic hardship (sum of 4 indicators: mother as single parent, maternal education less than high school, family poverty and family overcrowding), neighborhood threats, intensity of life events, maternal alcohol abuse, maternal depression, and poor quality of the home environment	<u>5 and 6 Years:</u> <i>externalizing and internalizing problem behaviors:</i> CBCL <i>cognitive performance:</i> Stanford-Binet at age 5, Weschler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R) at age 6	<p>Mediator(s) maternal depression and poor home environment as mediators of the relationship between other distal risks and child competence</p> <p>Moderator(s) home environment</p>	<p>adjusting for CI, but treatment did not modify the effect of CI. Sustained treatment effects at 5 and 8 years were only observed for children with moderate levels of human capital, but not for any levels of psychological adversities.</p>
							<p>Four different models were tested: independent-additive, CI, mediation, and interaction models – each as cross-sectional (age 5 risks and outcomes) and longitudinal (age 5 risks and age 6 outcomes).</p> <p>Independent-additive model: For age 5 cross-sectional analysis (adjusting for all other variables), economic hardship, neighborhood threats, intense negative life events were linear predictors of cognitive performance, and quality of home environment and intensity of life events were curvilinear predictors of cognitive performance. Economic hardship and intense life events predicted externalizing behaviors, and neighborhood threats and intense life events predicted internalizing behaviors. For age 6 longitudinal analysis (adjusting for age 5 outcomes), only cognitive performance at age 5 predicted performance at age 6. Maternal depression and age 5 externalizing behavior predicted age 6 externalizing behaviors, and quality of home environment, intensity of life events (squared), and age 5 internalizing behaviors predicted internalizing behaviors at age 6.</p> <p>CI model: Increased CI predicted worse outcomes for all three measures at age 5. At age 6, the effects of CI were not significant after controlling for age 5 outcomes.</p>

						<p>Mediation model: Maternal depression partially mediated the relationship between negative life events and externalizing problems at age 5. No mediators emerged for externalizing or internalizing problems at age 6. Quality of the home environment partially mediated the relationship between economic hardship and cognitive performance and age 5.</p> <p>Moderation model: In situations of high economic hardship, a high quality home environment protected children against externalizing behavior problems and low cognitive performance at age 6.</p>
Laucht, Esser & Schmidt, 1997	14	N=350	<p><u>At birth:</u> <i>biological risk:</i> defined by the degree of pre- peri- or neonatal complications; non-risk group was full-term, normal birth weight and no medical complications; moderate risk was pre-term but not complications; high-risk was low birth weight or clear asphyxia with special care treatment or neonatal complications.</p> <p><i>psychosocial index (11 items):</i> low parent education, crowded living conditions, parental psychiatric disorders, parental delinquency or broken home history, marital discord, early</p>	<p><u>3 Months, 2 Years and 4.5 Years:</u> <i>motor skills:</i> Psychomotor Developmental Index (PDI) of the Bayley Scales of Infant Development (at 3 mo and 2 years) and the Motor Quotient of the Test of Motor Abilities (at age 4.5 years)</p> <p><i>cognitive development:</i> Mental Developmental Index (MDI) of the Bayley Scales (at 3 mo and 2 years) and a composite of the Columbia Mental Maturity Scale the Illinois Test of Psycholinguistic Abilities, the Pictorial Test of Intelligence, and the Frostig Developmental Test of</p>	Covariates gender	<p>Increased biological risks were related to delayed motor development, impaired cognitive development, and worse social-emotional outcomes at all ages. Psychosocial index predicted motor delays as early as 2 years, and cognitive delays and social-emotional problems at all ages. Children with high biological risk and psychosocial index performed the least favorable on all outcomes at all ages. Whereas psychosocial index became more prominent with growing age and were related to poorer child outcome in all areas of functioning, biological risks decreased in influence and predominantly resulted in poorer motor development. With respect to individual predictors of cognitive outcomes at age 4.5 years, low parent education was the most significant, though nearly all psychosocial factors were associated with cognitive development. Of the biological factors, only pre-term birth, very low</p>

			parenthood, single-parent family, unwanted pregnancy, lack of social support, severe chronic difficulties, and poor coping skills; non-risk (score of 0), moderate risk (score of 1-2), high risk (score of 3 or greater)	Visual Perception (at 4.5 years)		birth weight, receipt of respiratory therapy after birth and seizures were associated with cognitive outcomes at ages 4.5. No significant gender differences.
				<i>social and emotional outcomes</i>		
Liaw & Brooks-Gunn, 1994	17	N=704	<p><u>0-1 Year:</u></p> <p><i>cumulative index (13 biological, socioeconomic, maternal, and family items):</i> birth weight, neonatal health status, race/ethnicity, maternal age, maternal education (all at birth), unemployment, maternal mental health, stressful life events, social support, father absence, and family density (at 12 mo), mothers score on verbal ability (PPVT; at 18 mo).</p> <p>CI measures collapsed into 5 groups for analysis (0-1, 2,3, 4-5, 6+)</p>	<p><u>3 Years:</u></p> <p><i>Stanford-Binet Intelligence Scale</i></p> <p><i>CBCL Problem Behaviors</i></p>	<p>Moderator(s)</p> <p>income-to-needs, receipt of intervention</p> <p>Covariates</p> <p>income to needs, receipt of intervention, child sex, clinical site</p>	<p>Poor families had a greater number of risks/adversities than non-poor families. All 13 independent variables explained 13% of variance of IQ above and beyond poverty, site and sex. Significant associations were seen with poverty status, race/ethnicity, maternal education, maternal PPVT and maternal depression. Family poverty status interacted with race/ethnicity and maternal PPVT; effects of being AA or Hispanic and having a mother with low PPVT were greater for poor children than non-poor children. For problem behaviors, independent exposures accounted for 12% of problem behaviors after accounting for covariates. Major contributors were mother PPVT, mother depression, stressful life events, and mother age at birth. No interaction effect of poverty. CI predicted child IQ. There was a significant interaction between CI and poverty; effect of CI similar for poor and non-poor children with fewer risks but poor children fared worse at high risks. Similar effect for problem behaviors, but no interaction. Regardless of poverty, children in the treatment group had higher IQ than did no treatment. Significant treatment x CI interaction for poor children only. Among poor children, the intervention had a greater effect for children who less exposures. IQs of</p>

						children who were poor and received the intervention were higher than for children who were not poor and did not receive the intervention. No treatment effect for behavior problems.
McFadden, Tamis-Lemonda, 2012	13	N=160	<u>15 months:</u> maternal depression, stressful life events, marital status at birth, maternal age at birth	<u>15 and 25 months:</u> <i>Bayley Scales of Infant Development (BSID-II):</i> memory, problem solving, early number concepts, generalization skills, classification abilities, vocalizations, language, and social skills	Mediator(s) <u>15 and 25 months:</u> <i>negative parenting:</i> composite of maternal negative affect, negative touch, and negative verbal statements <i>intrusive parenting:</i> composite of inflexibility, intrusiveness, and teasing <i>responsive/ didactic parenting:</i> positive affect, responsiveness to child, emotional attunement, achievement orientation, amount of language, quality of language, and symbolic play Covariates race/ethnicity, child age and gender	<p>Among low-income mothers, specific maternal characteristics predicted different aspects of parenting (i.e., young and stressed had more negative parenting whereas depressed were less responsive or intrusive; being married predicted all 3 parenting variables).</p> <p>Depression and responsiveness predicted infant cognition at 15 months; depression was mediated by responsiveness. Maternal characteristics did not predict child cognition over time (i.e. earlier measures did not predict cognition at 25 months), but responsiveness at 15 months predicted cog outcomes at 25 months. However, when responsiveness was low, intrusiveness played a buffering role; in such cases, intrusiveness may have provided children with stimulation that they otherwise were not receiving.</p> <p>Authors conclude that different adversities may result in different parenting behaviors. Different parenting behaviors may have unique effects on cog outcomes. Combinations of poor parenting behaviors may be particularly detrimental as opposed to only one poor parenting behavior.</p>
Poehlmann, 2005	13	N=60	<u>2-7 Years:</u> <i>caregiver sociodemographic</i>	<u>2-7 Years:</u> <i>IQ: Stanford-Binet</i>	Mediator(s)	Children of incarcerated mothers experienced multiple risks/adversities across contextual

CI, M	children (2-7 years) and their incarcerated mothers	<i>index (6 items):</i> less than high school education, currently single, having four or more dependents, currently unemployed, currently using public assistance, self-report of poor or fair health	Intelligence Scale; vocabulary, comprehension, pattern analysis, copying, and memory for sentences subscales	<u>2-7 Years:</u> <i>Quality of home and family environment:</i> HOME scale Covariates race/ethnicity <i>Incarcerated mother preincarceration cumulative index (6 items):</i> less than high school education, never married, having four or more children, gave birth to target child as a teenager, unemployed prior to incarceration <i>child biological risk index (3 items):</i> mother used substances during pregnancy, the child was born at 36 weeks gestation or less, the mother experienced complications during pregnancy, labor or delivery	levels. Caregiver index significantly predicted child cognitive outcomes, controlling for all other covariates. The home environment mediated this relationship. No other covariates were significantly associated with either the home score or child's cognitive outcomes.
	cross-sectional				
Sameroff, Seifer, Baldwin,	15	N=152	<u>4 and 13 Years:</u> <i>cumulative index (10 items):</i> minority group status,	<u>4 Years:</u> <i>Weschler Preschool and Primary Scale of Intelligence</i>	Covariates maternal IQ, race Risks/adversities were moderately stable over time. At age 4, all factors were independently associated with IQ. The strongest predictors

Baldwin & 1993	CI, T, Mod	families from Rochester Longitudinal Study; mostly white with oversampling of mentally ill families longitudinal	occupation of head of household, maternal education, family size, father absence, stressful life events (jobs, deaths, physical illness), parental perspectives, maternal anxiety, maternal mental health, mother-child interaction	(WPPSI): similarities, comprehension, information, vocabulary subscales <u>13 Years:</u> Revised WISC - information, similarities, picture arrangement, and block design subscales used	were race and social status domains (minority, head of household occupation, and mother education). Out of 10 risks, 7 were still significant to child IQ at age 13. Social status variables were the strongest, followed by parenting perspectives and maternal mental health. Life events, maternal anxiety and family interaction did not predict IQ. Both CI at age 4 and at age 13 predicted IQ (explained 50% of variance). Significance of CI remained after partialing out SES and minority status. Race moderated effect of CI on IQ at 4 years; blacks consistently had lower IQ whereas whites decreased with higher scores; there was less moderation at 13 years (i.e. same slope), but blacks still consistently lower than whites for equal amount of exposures. CI was a significant predictor of IQ after controlling for maternal IQ at both ages. Child IQ at age 4 was the biggest predictor of child IQ at age 13 (but correlation was only 0.35). Mother IQ and 4 yr CI were also significant predictors of 13 yr IQ. Magnitude of risk stability was the same as the IQ stability. Different clusters of risk factors did not produce difference in IQ.
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APPENDIX, Table A3.2, Studies of Multiple Adversities and Executive Function

C=Comparison study; CI=Cumulative Index; D=Domain; I=Intervention; Int=International; M=Mediation; Mod=Moderation; MR=Multiple Regression; T=Timing

Article	Score/ Codes	Sample & Design	Adversity Measure(s)	Cognitive & Other Outcomes	Mediating & Moderating Factors, Covariates	Findings
Brown, Ackerman & Moore, 2013	12 MR, D, T, M	N=120 children 3-5 years of age attending Head Start in Philadelphia longitudinal	<u>Time 1 (Fall):</u> <i>family income-to-needs ratio</i> <i>adversity index</i> : z-score of instability (the number of changes in where or with whom children had lived since their birth) and chaos (CHAOS scale) measures	<u>Time 1 (Fall):</u> PPVT-III Bracken Basic Concepts School Readiness Composite <u>Time 3 (Spring):</u> Bracken Basic Concepts School Readiness Composite	Mediator(s) <u>Time 1 (Fall), Time 2 (Winter), Time 3 (Spring):</u> <i>Inhibitory control</i> : Day/Night Stroop, Peg Tapping, Bear/Dragon assessments Covariates Child age, sex and verbal ability (PPVT)	Results show strong increases in inhibitory control over the preschool year with moderate stability within children. Family income did not relate significantly to inhibitory control in any assessment, and verbal ability related weakly to fall inhibitory control (measured concurrently) and not at all to future assessments. Adversity index related significantly to inhibitory control both concurrently and prospectively. Within the preschool year, the more unstable and chaotic the family, the smaller the growth in inhibitory control over time. Relationship between fall adversity index and spring school readiness was mediated by winter inhibitory control.
Evans & English, 2002	13 CI, M	N=287 children (8-10 years); 168 below poverty and 119 middle class cross-sectional	<u>8-10 Years:</u> <i>cumulative index (6 items): 3 psychosocial stressors</i> - exposure to violence, family turmoil, and child-family separation; 3 physical stressors - crowding, noise, and housing quality *Each indicator classified as risk if above 1 SD from the mean	<u>8-10 Years:</u> <i>psychological distress</i> : Rutter Children's Behavior Questionnaire (parental report) <i>self worth</i> : Global Self-Worth scale <i>self-regulation</i> : behavioral observations of self-regulatory behavior through a variant of the delayed gratification task <i>Psychophysiological stress</i> : 2 cardiovascular and 3	Mediator(s) CI as mediator between poverty and other outcomes Covariates poverty (income-to-needs), gender, maternal education, family composition, single-parent-status, age	Poor children were more often exposed to each stressor compared to non-poor children. Intensity of the stressor was also greater for poor children. Poor children also had a higher CI score on avg (2.8 vs 1.5). Poverty was significantly related to mental health and psychological distress, controlling for maternal education and single-parent status. Poverty also predicted delayed gratification and all biomarkers except NE. Poverty was strongly correlated to CI after controlling for maternal education and single-parent status. The only stressor metric that appeared to mediate effect of poverty on socioemotional development was the CI

				neuroendocrine markers of chronic stress (BP, creatine, unbound cortisol, epinephrine and NE)		metric (as opposed to individual or multiple risk approach).
Fearon & Belsky, 2004	16	N=918	<u>1, 6, 15, 24, 36 & 54 months:</u> <i>cumulative index (9 items; spanning psychological, social context and child factors):</i> income-to-needs ratio (avg. across all time points) father absence (frequency of single status across all time points), maternal depression (avg. between 6-54 mo), maternal social support (avg. across all time points), maternal age at birth, maternal education (1 mo only), maternal verbal IQ (PPVT; 36 mo), observed maternal support for cognitive development (avg. of 6,15, 36 & 54 mo), child difficult temperament (6 mo only) impulsivity (54 mo)	<u>4.5 Years:</u> <i>impulsivity:</i> CPT attention task <i>inattention:</i> CPT attention task <i>attention-related behavior problems:</i> CBQ and CBCL questionnaire	Moderator(s) child attachment: strange situation (15 months) Covariates gender	CI predicted all three attention-related outcomes such that higher CI was associated with more impulsivity, more inattention and more attention-related behavior problems. Male gender also predicted more behavior problems and impulsivity. For maternal reports of attention behavior, disorganized attachment scored significantly lower than other attachment categories, but there was no interaction between attachment, risk or gender. For the CPT inattention outcome, the avoidant and disorganized children scored generally higher in inattention, and particularly so for avoidant girls and disorganized children with greater CI. For CPT impulsivity, the avoidant group scored higher in impulsivity than did the secure group. Avoidant children showed higher impulsivity under high CI conditions. Authors conclude that children with secure attachment were less susceptible to the effects of CI and gender on CPT attentional performance than their insecure counterparts, and no differential risk susceptibility was evident for maternal reports of attention-related behavior.
Fishbein, Warner, Krebs, Trevarthen, Flannery & Hammond, 2009	14	N=553	<u>Age 10-12:</u> <i>any abuse:</i> Childhood Trauma Questionnaire <i>any neglect:</i> Childhood Trauma Questionnaire <i>school stress (made up of 5-items):</i> trouble with teacher,	<u>Age 10-12:</u> <i>Ravens Coloured Progressive Matrices:</i> nonverbal measure of general intelligence <i>Cambridge Decision-Making Task</i> (risk-taking)	Covariates child age and parent level of education	Bivariate relationships of stressors with cognitive outcomes showed higher levels of each stressor related to worse behavioral regulation. Abuse significantly related to lower Stop-Change impulsivity scores, and neglect significantly related to lower Raven's IQ and Ekman's facial recognition. In multi-variate regression (adjusting for all other stressors

			schools	getting suspended, being bullied	Tower of London Test: procedural memory and problem solving				and covariates), only neglect was significantly associated misattributions of emotion during the facial recognition task, and physical abuse was related to lower problem solving scores. All stressors except neglect and neighborhood stressors were related to behavioral regulation. Authors conclude that stressor types are differentially associated with different neurocognitive tasks and future studies should take into account these much more nuanced and complex relationships.
			cross-sectional	parent stress (made up of 7 items): fighting, parent involvement, helping with homework, feeling that they cared witnessing neighborhood violence perception of problems within neighborhood	Stroop Color Word Test: flexibility and resistance to interference Logan Stop-Change Task: inhibition that involves ability to shift responses in light of new information Ekman Facial Recognition Test: accurately identify emotional expressions Dysregulation Inventory: emotional, behavioral, and cognitive dysregulation; only used impulsivity subscale in current study; measure of ADHD symptoms				
Lengua, Honorado, Bush & 2007	13	N=80		<u>33-40 Months:</u> cumulative index (9 demographic and social factors): poverty, single-parent status, ethnic or racial minority status, household density, negative life events, parental depression, and history of mental health or legal problems	<u>33-40 Months:</u> effortful control (EC) composite of 5 tasks: 4 Stroop-like tasks (Bear-dragon, Day-Night, Grass-snow, Butterfly), and one delay of gratification task (using gift delay)	Mediator(s)			EC was moderately stable across 6 months. EC at time 1 was unrelated to concurrent CI or parenting behaviors. However, EC at time 2 was predicted by CI at time 1, controlling for EC at time 1. Maternal limit setting and scaffolding emerged as mediators of the effect of CI on EC at time 2. Social competence was predicted by EC at time 1, CI, and maternal warmth. Authors conclude that contextual risk predicts effortful control and aspects of parenting accounts for this effect.
			CI, M, T	preschool children (ages 33-40 mo), mostly white, middle class		<u>33-40 months:</u> maternal warmth, negative affect, limit setting, scaffolding: 4 measures assessed from restricted play, unrestricted free play and lego-building			
				longitudinal	Scores above 1.5 SD from the sample mean were considered "high risk"	<u>6 Months Later:</u> effortful control composite same as above) social competence: parent report of the Social Skills rating Scale (cooperation, assertion, responsibility, and	Covariates Verbal ability		

				self-control)	(PPVT) at 33-40 mo as a measure of general intelligence	
					child age, sex	
Mistry, Benner, Biesanz, Clark & Howes, 2010	19	N=1851	<u>T1 (0-12 mo) and T3 (24-36 mo):</u> <i>cumulative index (7 items):</i> maternal marital status, employment, income-to- needs, receipt of public assistance, maternal depressive symptoms, ability to meet basic needs, ability to meet medical needs *calculated as a proportion score because some families were missing some of the variables	<u>T3 (24-36 mo):</u> <i>cognitive/academic achievement (5-item latent construct):</i> WJ Psycho-Educational Battery-Revised (competencies in reading and math), WJ Letter-Word subscale, WJ Applied Problems subscale, PPVT, CAP Early Childhood Diagnostic Instrument (book knowledge subscale) <i>attentional/behavioral regulation (3-item latent construct):</i> sustained attention, engagement of parent, quality of play <i>problematic social behavior (3-item latent construct):</i> aggression, hyperactivity, withdrawn	Mediator(s) <u>T2 (0-12 mo) and T3 (24-36 mo):</u> maternal warmth/ responsiveness (HOME subscale) language/literacy stimulation in the home (HOME subscale) Moderator(s) race/ethnicity, Head Start enrollment Covariates child overall cognitive functioning (Bayley Scales), maternal education, gender	CI during infancy (T1) was most detrimental for all three outcome domains and was partially mediated by later CI, maternal warmth, and cognitive stimulation. The association between CI at infancy (T1) and preschool (T3) was strong, but not absolute, indicating some degree of instability. Findings suggest that timing of exposures in infancy is important compared to later years. No differences observed by race/ethnicity or early head start enrollment.
Raver, McCoy, Lowenstein & Pess, 2013	12	N=391	<u>Fall of Head Start Year (T), T+1, T+4:</u> <i>income to needs ratio</i> <i>school quality measures:</i> school-level poverty (free	<u>Fall of Head Start Year (T):</u> <i>early executive control:</i> composite from the Preschool Self Regulation Assessment, including Balance Beam and Pencil	Covariates child age, gender, race and intervention receipt	Early executive control difficulties and lower family income predicted later executive control difficulties in the 2 nd and 3 rd grades. Children with initial low levels of executive control were most vulnerable to deficits in later executive control within unsafe school climates. Boys and AA also had greater difficulties in

8 years) in Chicago School Readiness Project	lunch), school-level achievement, unsafe school climate, low adult support	Tap tasks	executive control. Findings suggest long-term effects of family adversity through elementary school transition, and school context should also be considered a potential adverse exposure.
longitudinal	<i>family measures:</i> single caregiver marital status, less than high school education	<u>T+4:</u> <i>executive control:</i> teacher report of Barratt Impulsiveness Scale and the Behavior Rating Inventory of Executive Function (BRIEF)	

REFERENCES

1. Nelson CA, de Hann M, Thomas KM. *Neuroscience of Cognitive Development: The Role of Experience and the Developing Brain*. Hoboken, New Jersey: John Wiley & Sons Inc; 2006.
2. Nisbett RE, Aronson J, Blair C, et al. Intelligence: New findings and theoretical developments. *American Psychologist* 2012;67:130–59.
3. Flouri E, Mavroveli S, Panourgia C. The role of general cognitive ability in moderating the relation of adverse life events to emotional and behavioural problems. *British Journal of Psychology* 2012;104:130–9.
4. Blair C, Zelazo PD, Greenberg MT. The Measurement of Executive Function in Early Childhood. *Developmental Neuropsychology* 2005;28:561–71.
5. Blair C. How similar are fluid cognition and general intelligence? A developmental neuroscience perspective on fluid cognition as an aspect of human cognitive ability. *Behavioral and Brain Sciences* 2006;29:109–60.
6. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci* 2009;10:434–45.
7. Pechtel P, Pizzagalli DA. Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology* 2010;214:55–70.
8. Brooks-Gunn J, Duncan GJ. The effects of poverty on children. *The Future of Children* 1997;7:55–71.
9. Anda RF, Felitti VJ, Bremner JD, et al. The enduring effects of abuse and related adverse experiences in childhood. *European archives of psychiatry and clinical neuroscience* 2006;256:174–86.
10. Sameroff AJ, Seifer R, Baldwin A, Baldwin C. Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development* 1993;64:80–97.
11. Evans GW, Li D, Whipple SS. Cumulative risk and child development. *Psychological Bulletin* 2013;139:1342.
12. Bethell CD, Newacheck P, Hawes E, Halfon N. Adverse Childhood Experiences: Assessing The Impact On Health And School Engagement And The Mitigating Role Of Resilience. *Health Affairs* 2014;33:2106–15.
13. Evans GW, Kim P. Multiple risk exposure as a potential explanatory mechanism for the socioeconomic status-health gradient. *Annals of the New York Academy of Sciences* 2010;1186:174–89.

14. Rutter M. Protective factors in children's responses to stress and disadvantage. *Annals of the Academy of Medicine, Singapore* 1979;8:324.
15. Sameroff AJ, Seifer R, Barocas R, Zax M, Greenspan S. Intelligence quotient scores of 4-year-old children: social-environmental risk factors. *Pediatrics* 1987;79:343–50.
16. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine* 1998;14:245–58.
17. Centers for Disease Control and Prevention. Adverse childhood experiences reported by adults---five states, 2009. *MMWR Morbidity and mortality weekly report* 2010;59:1609.
18. Anda RF, Butchart A, Felitti VJ, Brown DW. Building a Framework for Global Surveillance of the Public Health Implications of Adverse Childhood Experiences. *American Journal of Preventive Medicine* 2010;39:93–8.
19. Shonkoff JP, Boyce WT, McEwen BS. Neuroscience, molecular biology, and the childhood roots of health disparities. *JAMA: The Journal of the American Medical Association* 2009;301:2252–9.
20. McEwen BS. Early life influences on life-long patterns of behavior and health. *Ment Retard Dev Disabil Res Rev* 2003;9:149–54.
21. McEwen BS, Gianaros PJ. Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences* 2010;1186:190–222.
22. Bosch NM, Riese H, Reijneveld SA, et al. Timing matters: Long term effects of adversities from prenatal period up to adolescence on adolescents' cortisol stress response. *The TRAILS study. Psychoneuroendocrinology* 2012;37:1439–47.
23. Alaimo K, Olson CM, Frongillo EA. Food insufficiency and American school-aged children's cognitive, academic, and psychosocial development. *Pediatrics* 2001;108:44–53.
24. Ayoub C, O'Connor E, Rappolt-Schlichtmann G, Vallotton C, Raikes H, Chazan-Cohen R. Cognitive skill performance among young children living in poverty: Risk, change, and the promotive effects of Early Head Start. *Early Childhood Research Quarterly* 2009;24:289–305.
25. Aro T, Poikkeus A-M, Eklund K, et al. Effects of Multidomain Risk Accumulation on Cognitive, Academic, and Behavioural Outcomes. *Journal of Clinical Child & Adolescent Psychology* 2009;38:883–98.
26. Burchinal MR, Roberts JE, Hooper S, Zeisel SA. Cumulative risk and early

cognitive development: A comparison of statistical risk models. *Developmental Psychology* 2000;36:793–807.

27. Burchinal M, Vernon-Feagans L, Cox M, Key Family Life Project Investigator. Cumulative Social Risk, Parenting, and Infant Development in Rural Low-Income Communities. *Parenting: Science and Practice* 2008;8:41–69.
28. Hall JE, Sammons P, Sylva K, et al. Measuring the combined risk to young children's cognitive development: An alternative to cumulative indices. *British Journal of Developmental Psychology* 2010;28:219–38.
29. Klebanov PK, Brooks-Gunn J. Cumulative, Human Capital, and Psychological Risk in the Context of Early Intervention: Links with IQ at Ages 3, 5, and 8. *Annals of the New York Academy of Sciences* 2006;1094:63–82.
30. Krishnakumar A, Black MM. Longitudinal predictors of competence among African American children: The role of distal and proximal risk factors. *Journal of Applied Developmental Psychology* 2002;23:237–66.
31. Liaw F-R, Brooks-Gunn J. Cumulative familial risks and low-birthweight children's cognitive and behavioral development. *Journal of Clinical Child Psychology* 1994;23:360–272.
32. Poehlmann J. Children's family environments and intellectual outcomes during maternal incarceration. *Journal of Marriage and Family* 2005;67:1275–85.
33. Hooper SR, Burchinal MR, Roberts JE, Zeisel S, Neebe EC. Social and family risk factors for infant development at one year: An application of the cumulative risk model. *Journal of Applied Developmental Psychology* 1998;19:85–96.
34. Biederman J, Milberger S, Faraone SV, et al. Family-environment risk factors for attention-deficit hyperactivity disorder: a test of Rutter's indicators of adversity. *Archives of General Psychiatry* 1995;52:464.
35. Crozier JC, Barth RP. Cognitive and academic functioning in maltreated children. *Children & Schools* 2005;27:197–206.
36. Fishbein D, Warner T, Krebs C, Trevarthen N, Flannery B, Hammond J. Differential Relationships Between Personal and Community Stressors and Children's Neurocognitive Functioning. *Child Maltreatment* 2009;14:299–315.
37. McFadden KE, Tamis-LeMonda CS. Maternal Responsiveness, Intrusiveness, and Negativity During Play with Infants: Contextual Associations and Infant Cognitive Status in A Low-Income Sample. *Infant Ment Health J* 2012;34:80–92.
38. Brown ED, Ackerman BP, Moore CA. Family adversity and inhibitory control for economically disadvantaged children: Preschool relations and associations with school readiness. *Journal of Family Psychology* 2013;27:443–52.

39. Laucht M, Esser G, Schmidt MH. Developmental outcome of infants born with biological and psychosocial risks. *Journal of Child Psychology and Psychiatry* 1997;38:843–53.
40. Mistry RS, Benner AD, Biesanz JC, Clark SL, Howes C. Family and social risk, and parental investments during the early childhood years as predictors of low-income children's school readiness outcomes. *Early Childhood Research Quarterly* 2010;25:432–49.
41. Lengua LJ, Honorado E, Bush NR. Contextual risk and parenting as predictors of effortful control and social competence in preschool children. *Journal of Applied Developmental Psychology* 2007;28:40–55.
42. Pasco Fearon RM, Belsky J. Attachment and Attention: Protection in Relation to Gender and Cumulative Social-Contextual Adversity. *Child Development* 2004;75:1677–93.
43. Evans GW, English K. The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development* 2002;73:1238–48.
44. Cybele Raver C, McCoy DC, Lowenstein AE, Pess R. Predicting individual differences in low-income children's executive control from early to middle childhood. *Developmental Science* 2013;16:394–408.
45. Brown ED, Ackerman BP. Contextual Risk, Maternal Negative Emotionality, and the Negative Emotion Dysregulation of Preschool Children From Economically Disadvantaged Families. *Early Education & Development* 2011;22:931–44.
46. Ford DC, Merrick MT, Parks SE, et al. Examination of the factorial structure of adverse childhood experiences and recommendations for three subscale scores. *Psychology of Violence* 2014;4:432–44.
47. Gunnar MR, Barr RG. Stress, early brain development, and behavior. *Infants & Young Children* 1998;11:1–14.
48. De Bellis MD. Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology* 2001;13:539–64.
49. Carneiro P, Meghir C, Parey M. Maternal Education, Home Environments, and the Development of Children and Adolescents. *Journal of the European Economic Association* 2012;11:123–60.
50. Centers for Disease Control and Prevention. Essentials for Childhood: Steps to create safe, stable, nurturing relationships and environments 2014. Retrieved 2015 Sept 8. Available from: http://www.cdc.gov/violenceprevention/pdf/essentials_for_childhood_framework.

pdf.

51. Guo G, Harris KM. The mechanisms mediating the effects of poverty on children's intellectual development. *Demography* 2000;37:431–47.
52. Yeung WJ, Linver MR, Brooks-Gunn J. How money matters for young children's development: Parental investment and family processes. *Child Development* 2002;73:1861–1879.
53. Gershoff ET, Aber JL, Raver CC, Lennon MC. Income Is Not Enough: Incorporating Material Hardship Into Models of Income Associations With Parenting and Child Development. *Child Development* 2007;78:70–95.
54. Schoon I, Jones E, Cheng H, Maughan B. Family hardship, family instability, and cognitive development. *Journal of Epidemiology and Community Health* 2012;66:716–22.
55. Gunnar M, Quevedo K. The Neurobiology of Stress and Development. *Annu Rev Psychol* 2007;58:145–73.
56. McEwen BS. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology* 2008;583:174–85.
57. Evans G, Kim P, Ting A, Tesher H, Shannis D. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology* 2007;43:341.
58. Lupien S, King S, Meaney M, McEwen B. Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology* 2001;13:653–76.
59. De Bellis MD. The Psychobiology of Neglect. *Child Maltreatment* 2005;10:150–72.
60. Watts English T, Fortson BL, Gibler N, Hooper SR, De Bellis MD. The psychobiology of maltreatment in childhood. *Journal of Social Issues* 2006;62:717–36.
61. Repetti RL, Taylor SE, Seeman TE. Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin* 2002;128:330.
62. Huizink AC, Mulder EJH. Maternal smoking, drinking or cannabis use during pregnancy and neurobehavioral and cognitive functioning in human offspring. *Neuroscience & Biobehavioral Reviews* 2006;30:24–41.
63. Aarnoudse-Moens CSH, Weisglas-Kuperus N, van Goudoever JB, Oosterlaan J.

Meta-Analysis of Neurobehavioral Outcomes in Very Preterm and/or Very Low Birth Weight Children. *Pediatrics* 2009;124:717–28.

64. Forns J, Torrent M, Garcia-Esteban R, et al. Longitudinal association between early life socio-environmental factors and attention function at the age 11 years. *Environmental Research* 2012;117:54–9.
65. Roisman GI, Fraley RC. Developmental Mechanisms Underlying the Legacy of Childhood Experiences. *Child Dev Perspect* 2013;7:149–54.
66. Mendl M. Performing under pressure: stress and cognitive function. *Applied Animal Behaviour Science* 1999;65:221–44.
67. Taylor SE, Way BM, Seeman TE. Early adversity and adult health outcomes. *Development and Psychopathology* 2011;23:939–54.
68. Shonkoff JP, Garner AS, The Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood, Adoption, and Dependent Care, and Section on Developmental and Behavioral Pediatrics, et al. The Lifelong Effects of Early Childhood Adversity and Toxic Stress. *Pediatrics* 2011;129:e232–46.
69. Paulsell D, Avellar S, Martin ES, Del Grosso P. Home visiting evidence of effectiveness review: Executive summary 2010; Retrieved 2015 Sept 8; Available from: http://homvee.acf.hhs.gov/HomVEE_Executive_Summary.pdf.
70. Camilli G, Vargas S, Ryan S, Barnett WS. Meta-analysis of the effects of early education interventions on cognitive and social development. *Teach Coll Rec* 2010; 112:579-620.

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CHAPTER 4

TIMING OF MULTIPLE ADVERSE EXPERIENCES AND CHILD COGNITIVE DEVELOPMENT

INTRODUCTION

Significant stress or adversity in early life can impair child cognitive performance (1,2), including general cognitive ability (i.e., intellectual capacity, IQ) and executive functions (i.e., higher-order cognitive abilities including sustained attention, working memory and impulse control) (2-4). Although the detrimental effect of poverty on children's cognitive development is well documented (5), other adversities that disrupt the safety, stability and nurturance of a child's environment (i.e., abuse, neglect, family instability, parental mental illness, parental substance abuse, parental incarceration, domestic violence and neighborhood violence) also influence cognitive outcomes (6-9). These adversities often co-occur (10), and exposures to multiple adverse experiences is associated with worse developmental outcomes (7). The current study examines the influence of multiple adverse experiences on measures of child attention, working memory and verbal ability, which are critical cognitive outcomes for future health and achievement (11). We build upon the current literature by focusing on the influence of different domains, or types, of adverse exposures as well as the role of timing of exposures in relation to these outcomes.

Cumulative Adversities

Rutter and Sameroff were the first to show that children exposed to multiple adverse experiences had worse cognitive outcomes relative to children with any single adverse exposure (8,12). The well-publicized Adverse Childhood Experiences (ACE) Study also showed an association between adults' recollection of the number of adverse childhood experiences they experienced prior to age eighteen and numerous health risk

behaviors and diseases in adulthood (13). Since the ACE Study, evidence has accumulated to support the inverse relationship between multiple adverse exposures in childhood and health and developmental outcomes across the lifespan, including cognitive outcomes and achievement (7). This gradient has been observed from infancy through adolescence for outcomes of general cognitive ability (8,14-24), and executive function (25-28).

Studies (including the ACE study) commonly use a cumulative index metric, also known as “cumulative risk,” to examine the relationship between multiple adverse exposures and developmental outcomes. To construct this metric, adversities are dichotomized into exposed and unexposed categories and then summed to create an aggregate score (see Evans, Li and Whipple, 2013, for an in-depth discussion of this approach). In the current paper, we refer to this metric as a *cumulative adversity* index rather than the more commonly used *cumulative risk* index in order to distinguish adverse exposures from other risk factors. We define adverse exposures as *experiences that typically create excessive demands or threats to the child but are preventable or amenable to change*, thus lending themselves to intervention. Risks, on the other hand, refer to other factors, such as genetic predispositions or birth complications.

A strength of the cumulative index is that it is easily understood and communicated to laypersons and policy makers (7,25). Additionally, this method preserves statistical power in small sample sizes and avoids issues of collinearity by combining multiple, highly correlated measures into a single metric (7). However, the cumulative index also weights each adversity equally, and this limits the ability to make inferences about the salience of specific adverse exposures or the relations between

exposures, making it difficult to inform the most effective targets for interventions. As a result, some have criticized this method as atheoretical (7).

Adversity Domains

Evans, Li and Whipple (2013) proposed the use of domains as an alternative to a cumulative index (7). Domains are created by aggregating adversities of a similar type or context into a number of groups (7). For example, an economic hardship domain could be formed by aggregating measures of poverty, food insecurity and housing insecurity. A domain-based approach is promising in that it leverages the advantages of a cumulative measure while also providing additional insight into the salience of particular domains or relations between domains (7). Consequently, a domain-based approach to the study of multiple adversities also allows for more theoretically driven models linking adverse exposures to developmental outcomes.

While there is no current consensus on the best formulation of adversity domains, we propose three domains based on our prior review of the literature in Chapter 3 that examined the different types of adverse exposures known to influence cognitive development. These are 1) economic hardship, 2) family instability, and 3) lack of safety. We conceptualize economic hardship as living at or below the federal poverty level and lacking the financial resources to cover basic needs, including food and housing. Family instability, refers to the characteristics of families that challenge the continuity or predictability of the home environment (29). We conceptualized this domain to include factors such as the stability of parental relationships, parental incarceration, maternal depression or parental substance abuse. The third domain includes exposures that threaten

a child's physical safety, including exposure to abuse, neglect, domestic violence or community violence.

From a theoretical perspective, economic hardship is thought to influence child cognitive development not only through lack of material resources available to invest in a child's stimulation, nutrition, physical environment and health (5,30), but also because the stress of being unable to make ends meet interferes with the ability of parents to allocate time and energy to interact positively with their children (31). Unstable and chaotic home environments are closely linked with poverty and may also disrupt cognitive development by interfering with parent/child interactions (32). All three domains influence cognitive development through disruptions in stress neurobiology (2,33-37). Under conditions of stress, a coordinated physiological response enables an individual to adapt to the threat (1). The extensively studied hypothalamic-pituitary-adrenal (HPA) axis is central to this process, resulting in the production of cortisol that serves to regulate the stress response (1,38,39). However, severe or chronic stress that persists over time can lead to over-active or deregulated responses – both associated with alterations in brain structure and function that impair cognitive development (1,37,39).

Although there is sufficient evidence to suggest that both general cognitive ability and executive functions are negatively influenced by economic hardship (5,40), family instability (41), and lack of safety (2,33,38), fewer studies have explored the effects of these domains simultaneously. Klebanov and Brooks-Gunn (2006) compared a human capital domain (a cumulative score of maternal employment, education and welfare status) and a psychological domain (a cumulative score of low monetary, emotional and child care support, maternal depression, and stressful life events) – both based on

measures assessed in infancy (19). Although higher scores in both domains separately predicted worse general cognitive ability at ages three, five and eight years using simple regression, only the human capital domain predicted worse general cognitive scores at all three ages after accounting for both domains simultaneously. This is consistent with the results from another longitudinal study that showed no significant effects of family instability on cognitive functioning in five year olds, after controlling for economic hardship and other family characteristics assessed across early childhood (32). Others showed that measures of family dynamics, including chaos and instability, explained the variance in inhibitory control, a measure of executive function, among a low-income sample of children, whereas poverty did not (41). These findings suggest that different adversity domains may differentially influence cognitive outcomes. Therefore, studies of multiple adverse exposures that rely only on a cumulative index may overlook the relative importance of certain types of adversities.

Timing of Adverse Exposures

The timing of adverse exposures may also influence cognitive outcomes. Neuroimaging studies show that brain development occurs throughout childhood and adolescence, with different regions of the brain developing at different times (1). Experience shapes this development (3). Neuroplasticity, or the molding of the brain in response to environmental input, is thought to facilitate adaptation to one's environment (42). Rapid development of specific brain regions during childhood and adolescence create windows of opportunity when these brain regions are more sensitive to experience – positive or negative (1). Intense or prolonged exposure to “toxic stress” is thought to

impair specific brain regions with a high density of glucocorticoid receptors, including the hippocampus, amygdala and prefrontal cortex – regions of the brain that detect and respond to conditions of stress and are also associated with memory, learning and attention (1,43,44). Given that these brain regions develop most rapidly at different periods across childhood and adolescence, vulnerability to the effects of chronic stress may depend on the timing of exposure (1). For example, the hippocampus, known to influence memory, develops rapidly from birth to age two, and therefore, earlier adverse exposure during this window may be particularly detrimental (1). On the other hand, the protracted development of the prefrontal cortex, associated with the maturation of executive function, may be more vulnerable to the effects of chronic stress in adolescence (1).

Longitudinal studies of multiple adverse exposures and cognitive outcomes have shown that adversities are moderately to highly stable over time (8,17) making it difficult to disentangle the effects of sensitive periods of exposure from the effects of persistent or chronic exposures. However, there is some evidence to suggest that exposure to adversity in early childhood has lasting effects on cognitive outcomes (19,25). Additionally, some have shown that disparities in cognitive outcomes between children with high and low levels of early adverse exposures tend to increase as children age (15,17). General cognitive ability is thought to be moderately to highly stable across development, but children with a greater number of adverse exposures show declines in cognitive ability over time compared to children with less adversity in their lives (15,17). Executive functions, on the other hand, improve throughout childhood for all youth, tracking with the development of the prefrontal cortex (45). Studies have showed that

children exposed to multiple adversities show less improvement in executive function over time (41,46). Others have shown that although disparities in executive function are associated with early exposure to poverty, the gap between those who begin life in poverty compared to more affluence does not increase over time (47). Therefore, the effect of timing of adversity exposure is not yet clear. More longitudinal research is needed to discern *when* children may be particularly vulnerable to multiple adverse exposures. This is essential for informing the timing of interventions.

The Current Study

The current study builds on this body of work, drawing upon publically available data from a longitudinal cohort of children in the Fragile Families and Child Wellbeing Study. We examined the influence of multiple adversities (formulated as both a cumulative index and the above mentioned adversity domains) and their timing on pre-school and late childhood cognitive outcomes. Adversities were measured when children were very young (at infancy and around three years old), in the pre-school phase (five years) and late childhood (nine years), and cognitive outcomes were assessed at ages five and nine years. We hypothesized that a higher number of total adversities as well as higher scores in each adversity domain would predict worse cognitive outcomes. Furthermore, we hypothesized that exposure to adversity when children were very young (at infancy and age three) would directly predict cognitive outcomes at ages five and nine years, even after controlling for concurrent adverse exposures, demonstrating that early exposures were more influential. Taking further advantage of the nature of longitudinal research, we also examined whether the exposure to the different adversity domains

during the preschool years mediated the relation between early exposure to the adversity domains during infancy and cognitive outcomes at ages five and nine.

METHODS

Sample

The Fragile Families and Child Wellbeing (FFCW) Study follows a birth cohort of 4789 children born between 1998 and 2000 from 20 large U.S. cities (population >200,000) (48). The sampling of individuals occurred in three stages: first cities, then hospitals within cities, then births within hospitals. Children born to unmarried parents were oversampled ($n=3647$ vs. $n=1141$ children born to married parents) in order to be representative of non-marital births (see Reichman, Teitler, Garfinkel, & McLanahan for more details on study design).

As part of the core study, biological mothers were interviewed in the hospital within 48 hours of the focal child's birth, and biological fathers were interviewed by phone soon after. Both biological parents were interviewed again by phone when the child was one, three, five and nine years of age. The current paper also draws upon the In-Home Longitudinal Study of Preschool-Aged Children, a sub-study in which biological mothers who participated in the core study at years three and five were invited to participate. For the sub-study, primary caregivers participated in an additional interview and a home visit when the child was three and five years old. During the home visit, an investigator observed the home environment and directly assessed the child. At age nine, the in-home sub-study was integrated with the core study such that all

participants started with a home visit and then completed the primary caregiver and core surveys.

From the original 20-city sample (N=4789), 132 (3%) of families were ineligible for the current study because children had conditions likely to influence cognitive outcomes, including: total or partial blindness, total or partial deafness, Down's syndrome, cerebral palsy, mental retardation or other developmental delay, and autism. Additionally, 1391 (29%) families were excluded because the child did not have at least one cognitive outcome measurement at age nine. To minimize measurement bias, the sample was also limited to those in which the biological mother was the primary caregiver (as opposed to father or other guardian; 290 excluded (6%)). The final analytic sample (N=2976) was slightly more advantaged than those excluded from the final analysis (35% below federal poverty line vs. 39%; 37% of mothers with greater than high school education vs. 31%), more likely to be non-Hispanic black (50% vs. 43%) and less likely to be non-Hispanic white (20% vs. 23%), Hispanic (27% vs. 29%), or other race (3.4% vs. 5.3%). Forty percent of mothers were not married or cohabitating at the time of the child's birth, consistent with the oversampling of non-marital births planned for in the study design. However, given the differences between the original and analytical samples, the final analytical sample can no longer be considered representative of non-marital births in large U.S. cities. Descriptive characteristics of the final sample are shown in Table 4.1.

Measures

Cognitive Outcomes. The FFCW study collected well-established measures of cognitive functioning in children (49). The following assessments were conducted with the focal child in the child's home by a field interviewer during the in-home assessments.

Wechsler Intelligence Scale for Children (WISC-IV), Digit Span subtest. The WISC-IV is an intelligence test for children ages 6-16 years designed to measure child cognitive function. The Digit Span subtest of the WISC-IV specifically measures the child's auditory short-term memory, sequencing skills, attention, and concentration. At age nine, children heard a sequence of numbers and were asked to repeat the numbers either forward or backwards. Scores were age-normed (standard score of $M=10$, $SD=3$). The subtest has high internal consistency ($\alpha = 0.92$) and high test-retest reliability ($r = 0.89$) (50).

Child Peabody Picture Vocabulary Test (PPVT)-III. The PPVT-III measures receptive vocabulary and screens for verbal ability. At ages five and nine, an interviewer read a word and asked the child to identify the corresponding picture (among a set of four pictures) on an easel. Scores were age-normed (standard score of $M = 100$, $SD = 15$). The PPVT-III has high internal reliability ($\alpha = 0.93$) and test-retest reliability ($r=0.95$) (51).

Leiter International Performance Scale —Revised, Sustained Attention and Lack of Impulsivity. The Leiter International Performance Scale —Revised measures children's ability to maintain attention to a specific stimulus over time. At age five, children were shown a picture booklet with a variety of objects placed throughout the page. There was a target object at the top of the page, and children were asked to put a

line through as many of the matching target pictures as possible within the allotted time, without erroneously crossing out non-target objects. Average performance across four trials yielded two attention scores. The number of correct responses reflected the child's *sustained attention* whereas the number of incorrect responses (reverse coded) reflected *lack of impulsivity*. Scores were age-normed (standard score of $M=10$, $SD = 3$). The task has high internal reliability ($\alpha = 0.83$) and test–retest reliability ($r = 0.85$) for children 4–5 years of age (52).

Adverse Experiences. Biological mothers reported on the following measures during the core and the primary caregiver interviews at baseline and ages one, three, five and nine. In the first year of life, some of the adversity measures were collected at either baseline or age one, and therefore, these waves were combined and collectively referred to as *infancy*. Where possible, the same adversity measures were used at each wave of data collection. However, as described below, there were a few instances where measures differed across waves, or where adversities were not measured at all waves. All of the adversities were dichotomized such that 1 = exposed, and 0 = unexposed based on theoretical cut-points.¹⁴

Severe Psychological Aggression. The Parent-Child Conflict Tactics Scales (PCCTS) measures child maltreatment and nonviolent modes of discipline by parents (53). The 5-item psychological aggression subscale of the PCCTS measures verbal and symbolic acts by the parent intended to cause a child psychological pain or fear. During the primary caregiver survey at ages three, five and nine, biological mothers were asked

¹⁴ Maternal drug and alcohol use were also explored as adverse exposures in this study. However, too few respondents in the sample (<1%) reported these exposures, and therefore, these measures were not included in the final analysis.

how often they had done the following to the child in the past year: shouted, yelled or screamed at; threatened to spank or hit but didn't actually do it; swore or cursed at; called him or her dumb, lazy or some other name like that; said they would send them away or kick them out of the house. Ordinal responses included "never," "once," "twice," three to five times," etc. Among national samples, approximately 90% of parents report one or more forms of psychological aggression (also reflected in the current study population) (54). However, more severe forms of aggression (swore or cursed at, called dumb or lazy, or threatened to kick out of the house) are less common. Children of mothers who reported that at least one of these *more severe acts* occurred at least once in the last year were categorized as exposed to psychological abuse (prevalence score cut-offs for all PCCTS measures described in Straus et. al., 1998).

Severe Corporal Punishment. Corporal punishment was assessed with a 5-item subscale of the PCCTS. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often in the past year they: spanked the child on the bottom with a bare hand; hit the child on the bottom with something like a belt, hairbrush, a stick or some other hard object; slapped the child on the hand, arm or leg; pinched the child; and shook the child. Ordinal responses included "never," "once," "twice," three to five times," etc. Spanking the child and slapping the child on the arm or leg are considered to be more widely accepted forms of corporal punishment, whereas the other three acts are considered to carry higher risks and be less widely accepted, thus indicating more severe corporal punishment (55). Children of mothers who reported that at least one of these three *more severe acts* occurred at least once in the last year were categorized as exposed to severe corporal punishment.

Child Neglect. Child neglect was assessed with a 5-item subscale of the PCCTS. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often they: had to leave the child home alone, even when they thought an adult should be with the child; were so caught up with her own problems that they were not able to show or tell the child they loved him/her; were not able to make sure that the child got the food he/she needed; were not able to make sure the child got to a doctor or hospital when needed; had drinking or drugs interfere with taking care of the child. Ordinal responses included “never,” “once,” “twice,” “three to five times,” etc. Children of mothers who reported that at least one of these acts occurred at least once in the last year were categorized as exposed to child neglect.

Intimate Partner Violence (IPV). During the core surveys when the child was one, three, five and nine, biological mothers were asked to think about their relationship with the child’s father, or their current partner. For each existing romantic relationship (either with the biological father or a current partner), they were asked previously validated questions (56,57): 1) How often does he slap or kick you?; 2) How often does he hit you with a fist or object that could hurt you?; and 3) How often does he try to make you have sex or do sexual things that you don’t want to? For any relationship with the father (romantic or not) as well as for existing relationships with another current partner, mothers were also asked, “Have you and the father (or current partner) been in a physical fight in front of the child in the time since the last interview?” If mothers answered “sometimes” or “often” to any of the first three questions, or, “yes” to the last question, they were categorized as experiencing IPV for that time period.

Exposure to Community Violence. Different measures were used to assess exposure to community violence at infancy and the later waves. At infancy, biological mothers were asked during the baseline core survey how safe the streets around their house were at night (very safe, safe, unsafe or very unsafe). Responses of unsafe or very unsafe were categorized as exposure. During the primary caregiver surveys at ages three, five and nine, biological mothers were asked about their own exposure to violence in their neighborhood in the past year. Three questions assessed whether the primary caregivers saw someone get hit, punched, slapped or beaten up by someone else; if they saw someone get attacked with a weapon like a knife or a bat; and if they saw someone get shot. Ordinal responses ranged from never to more than ten times. Exposure to community violence at these waves was defined as at least one exposure to any of these three items.

Parental Relationship Instability. Relationship instability was defined as a change in parental relationship status since the child's birth (58,59). Prior studies using data from the FFCW Study have shown that children with stable family structures (whether married, cohabitating, or single parents) had better outcomes than children with unstable family structures (characterized by a parent's partial presence) (60). During the core surveys at baseline and when the child was three, five and nine, biological mothers were asked about their relationship with the biological father. Responses were categorized into: married, cohabitating or single. During the infancy wave, adversity was simply classified as having a single parent family structure at the time of the child's birth, as opposed to a married or cohabitating family structure. For the remaining waves, stability was defined as having the same parent structure since the previous wave or

moving from a cohabitating relationship to a married relationship since the previous wave. Moving from a married relationship to a cohabitating or single status, or moving from cohabitating relationship to single status was categorized as unstable.

Maternal Depression. The Composite International Diagnostic Interview (CIDI) is a standardized instrument for assessing mental disorders based on DSM-IV criteria. The short form (SF) of the CIDI interview takes a portion of the full set of CIDI questions and generates from the responses the probability that the respondent would be a case, if given the full interview (61). When the child was one, three, five and nine, biological mothers were asked all of the essential CIDI-SF questions necessary to classify a major depressive episode. Mothers who met established criteria were classified as probable cases for maternal depression.

Father Incarceration. Father incarceration was determined from both the mother report on the core surveys when the child was one, three, five and nine, and from information collected by interviewers in the field. Mothers were asked whether the father was currently in jail. Fathers were categorized as currently in jail if mothers or interviewers indicated this to be the case.

Living in Poverty. The income to needs ratio adjusts family income by the number of adults and children in the household, using the official poverty thresholds. Absolute poverty is measured by having a poverty ratio less than one. Family income and family size were collected from the biological mother during the core survey at baseline and ages three, five and nine. Living in poverty was defined as living below the federal poverty level.

Housing Insecurity. During the core surveys at ages one, three, five and nine, biological mothers were asked four questions derived from the Survey of Income and Program Participation and the New York City Social Indicators Survey (62), including whether they: had been evicted from their home in the past 12 months; stayed in a shelter/car or abandoned vehicle; did not pay full rent or mortgage; or if they had moved in with other people because of financial problems. Mothers responding “yes” to at least one of these questions were categorized as experiencing housing insecurity.

Food Insecurity. During the core survey at age one, the primary caregiver survey at age three, and the core surveys at ages five and nine, biological mothers were asked about whether, in the past 12 months, they were ever hungry but could not afford to buy more food (62). Mothers who responded “yes” to this question were characterized as experiencing food insecurity.

Cumulative Adversity Index. A cumulative adversity index was created for infancy, and ages three, five and nine. Each index was constructed by summing the total number of adversity exposures for each wave. During infancy, eight adversity exposures were summed into a single cumulative index with a possible score ranging from 0-8 (exposure to community violence, maternal IPV, single parent, maternal depression, father incarceration, living in poverty, housing insecurity and food insecurity). Eleven adversities were measured at years three, five and nine, so possible scores range from 0-11. These were the same as the infancy wave, with the exception that parental relationship instability was used instead of single parent status, and the three PPCTS measures were included (severe psychological aggression, severe corporal punishment, and child neglect).

Adversity Domains. As discussed, grouping adverse exposures into domains allows analysis of potentially different effects of types of adversities. Three theoretically determined adversity domains, *lack of safety*, *instability* and *economic hardship*, were created for each wave of the study by grouping adversities of a similar context. The *lack of safety* domain in infancy included two adversities (exposure to community violence and maternal IPV), and this domain at ages three, five and nine each included five adversities (severe psychological aggression, severe corporal punishment, neglect, community violence and maternal IPV). The *instability* domain included three adversities at all four waves: exposure to parental relationship instability (or single parent status at infancy), maternal depression, and father incarceration. The *economic hardship* domain included the same three adversities at all four waves: living below the poverty level, food insecurity and housing instability. Each domain was created by summing the total number of adverse exposures within that domain.

Control Variables. During the baseline core survey, biological mothers reported on the following control variables: maternal race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, and other), child sex (male/female), maternal education (high school education or less vs. greater than high school education), and neonatal risk. Neonatal risk was a constructed variable denoting whether the child was born with low birth weight (<2500g) *or* as a part of a multiple birth (twin). Publicly available data from the FFCW Study recorded all children born as a twin as missing in birth weight. Since twins are more likely to be born low birth weight, the two variables were combined into a

single measure. Even if a child was part of a multiple birth, only one focal child was included in the study.

In addition to these variables, prenatal substance use (drinking, smoking, drug use), maternal age, and maternal cognitive ability (similarities subtest from the Wechsler Adult Intelligence Scale – Revised (63)) were also included as controls in preliminary analyses but not retained in the final analyses. The prenatal substance abuse variables and maternal age were dropped due to lack of significance in relation to all cognitive outcomes in both bivariate and multivariate analyses. Maternal cognitive ability was dropped because this variable was moderately correlated with maternal education ($r = 0.37$), and maternal education was used as a proxy for maternal cognitive ability.

Sensitivity analyses were conducted for all final models that included both maternal education and maternal cognitive ability, and there were no substantive differences in the relations between adversities and cognitive outcomes between the models that included both maternal education and cognitive ability and those that did not control for mother cognitive ability.

Missing Data Analysis

As shown in Table 4.1, data were missing for less than 1% of cases for each of the control variables, 38% - 44% for the age five cognitive outcomes, less than 1% for the age nine cognitive outcomes, 11% - 35% for the cumulative adversity indices, and 2% - 28% for the adversity domains (individual adversity variables were missing between 0% - 26%; not shown in table). Data were missing mostly due to attrition rather than item non-response. Missingness due to attrition was less than 7% for each of wave of the core

study, 24% for the age three primary caregiver interview, 26% for the age five primary caregiver interview, and 38% for the in-home cognitive assessments. In order to identify potential systematic reasons for missing data, the relations between observed variables and missing values were examined empirically (64). In general, those who participated in the core and primary caregiver interviews were more advantaged and less likely to be Hispanic compared to those who did not participate, consistent with earlier trends describing the analytic sample. However, those who participated in the in-home assessments were *less* advantaged than those who did not participate.

Missing data were classified as missing at random (MAR) because factors associated with missingness were observed in the data set (such as race, education, poverty level and marital status). While the MAR mechanism introduces bias, this bias is recoverable with modern missing data methods (65). We used full-information maximum likelihood (FIML) to handle missing data in these analyses. FIML is a model-based approach that uses all of the available data to estimate the parameters of the statistical model in the presence of missing data and produces unbiased estimates of model parameters and standard errors (65,66). In this sample, nearly all variables missing more than 5% of data were correlated with at least one other variable used in the model or one of nine auxiliary variables (variables that were peripheral to the substantive analysis but provided information about missingness) at a correlation greater than 0.30.

Analytic Approach

We first examined the means and standard deviations for continuous variables, frequencies for categorical or dichotomous variables, correlations between study

variables, and unadjusted and adjusted path models, regressing each cognitive outcome and all other variables (including individual adversities, cumulative adversity indices for each age, and adversity domains for each age) using in Mplus 7.3 (67). Linear assumptions between the cumulative adversity indices, adversity domains, and cognitive outcomes were examined graphically in Stata 13.

Hypotheses were analyzed using path analysis in Mplus 7.3 (67). Path analysis is a type of structural equation modeling that estimates a system of equations between observed exogenous (predicting) and endogenous (mediating or outcome) variables. Unlike latent variable models, path models assume no measurement error in observed variables. Path analysis distinguishes three types of effects between variables: a direct effect is the influence that one variable has on another that is not mediated by any other variable in the model; an indirect effect is the influence of one variable on another through mediation of at least one other variable; and the total effect is the sum of the direct and indirect effects (68). Path models were evaluated based on the model fit (CFI >0.9 indicates acceptable fit and >0.95 close fit; RMSEA <0.08 indicates acceptable fit and <0.05 close fit), significance of path estimates, and the explained variance among the endogenous variables (68,69). Z-scores ($M=0$, $SD=1$) were created for all cognitive outcomes and used in all analyses (aside from descriptive analyses) in order to facilitate comparisons across outcomes.

RESULTS

Preliminary Analyses

Means and standard deviations for continuous variables, and frequencies for categorical or dichotomous variables are presented in Table 4.1. As expected with a relatively disadvantaged sample, mean scores on all of the age five and age nine child cognitive assessments were at or just below the normed average. Mean cumulative adversity index scores were 1.4 (SD=1.6) for infancy, 2.1 (SD=3.0) at age three, 2.2 (SD=3.1) at age five, and 2.3 (SD=3.0) at age nine, consistent with others who have shown that U.S. children experience, on average, two adversities (10). Mean scores on the adversity domains ranged from 0.2-1.3 (SD=0.2-1.4) across waves for the lack of safety domain, 0.3-0.6 (SD=0.3-0.5) for the instability domain, and 0.6-0.7 (SD=0.5-0.6) for the poverty domain.

Correlations among all model variables are displayed in Table 4.2. Correlations were classified as low (<0.1), modest (0.1-0.3), moderate (0.31-0.5) and strong (>0.5). Cumulative indices were negatively correlated with all cognitive outcomes, as expected. Positive correlations between cumulative indices across waves ranged from moderate to strong, indicating stability in these constructs over time. All adversity domains were also negatively correlated with all cognitive outcomes, as expected. Positive correlations among adversity domains within waves were modest to moderate, all in the expected direction. Across waves, similar adversity domains were modestly to moderately correlated, all in the expected direction, indicating only moderate stability in adversity domains over time.

We also conducted univariate path models with each cognitive outcome regressed on each of the cumulative indices and adversity domains to examine the nature of these relations.¹⁵ Results for these models are shown in the Appendix, Table A4.3. At each age, higher cumulative index scores predicted lower scores on each cognitive outcome (all estimates significant at $p < 0.05$). For the adversity domains, higher economic hardship scores at each age predicted significantly lower scores for all cognitive outcomes. The lack of safety domain also predicted lower scores on each cognitive outcome, though the coefficient was not significant for the relation between lack of safety in infancy and lack of impulsivity, or the relation between lack of safety at age three and sustained attention. The instability domain only significantly predicted lower PPVT-III scores at ages five and nine. There were no significant effects of the instability domain on any of the attention measures.

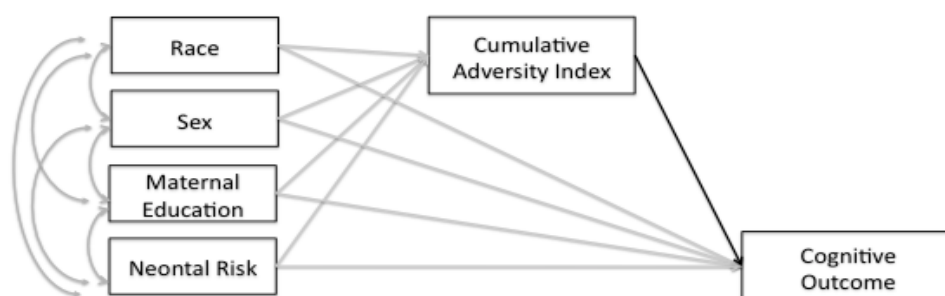
Relation Between Cumulative Indices and Cognitive Outcomes

To test the first hypothesis (2.1a) that *an increase in the total number of adverse exposures at each age would predict worse cognitive scores*, simple path models were examined with each cognitive outcome regressed on the cumulative adversity index for each age, one at a time, controlling for covariates. In these path models, both the cognitive outcome and cumulative adversity index were regressed on the set of covariates (shown in Figure 4.1). All models were a good fit ($RMSEA < 0.05$; $CFI > 0.95$). As

¹⁵ While the current study focused on whether the cumulative indices and adversity domains predicted the cognitive outcomes, our preliminary analysis also examined univariate path models between individual adversities and each cognitive outcome (Appendix, Table A4.1), followed by adjusted relations between each individual adversity and each cognitive outcome, controlling for covariates (Appendix, Table A4.2). In general, while many adversities from each wave were significantly associated with each cognitive outcome in the univariate path models, poverty was most consistently associated with each outcome in the adjusted models.

hypothesized, cumulative adversity scores were negatively associated with all cognitive outcomes after controlling for covariates, though not all coefficients were significant (results shown in the Appendix, Table A4.4). Among the age five cognitive outcomes, only the concurrent cumulative adversity index was associated with sustained attention ($\beta=-0.06$; $p<0.05$), none of the cumulative adversity indices were significantly associated with lack of impulsivity, and all of the cumulative adversity indices were significantly associated with the PPVT-III ($\beta=-0.10$ to -0.12 ; $p<0.001$). Among the age nine outcomes, the cumulative adversity indices at infancy ($\beta=-0.05$; $p<0.05$), age three ($\beta=-0.08$; $p<0.001$), and age nine ($\beta=-0.07$; $p<0.02$) were negatively associated with the age nine digit span, and all indices from all ages were associated with the age nine PPVT-III ($\beta=-0.06$ to -0.10 ; $p<0.001$).

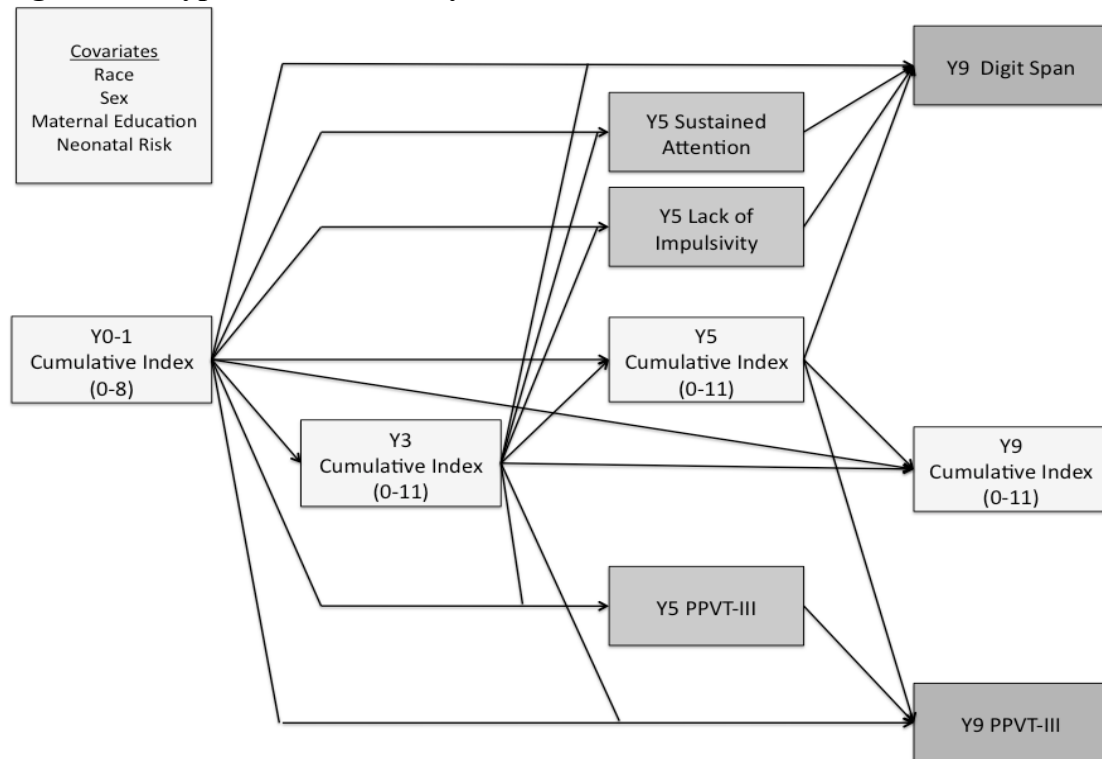
Figure 4.1 Hypothesis 2.1a Analytic Model



The conceptual model shown in Figure 4.2 was used to test the next hypothesis (2.1b) that *an increase in the total number of adverse exposures during infancy and at age three would directly predict worse cognitive scores at ages five and nine, after controlling for adverse exposures at ages five and nine*. The model was carefully constructed to monitor any change in variance of the cognitive outcomes with the

addition of each cumulative index measure from subsequent waves (model building results are shown in the Appendix, Table A4.5).

Figure 4.2. Hypothesis 2.1b Analytic Model ^a



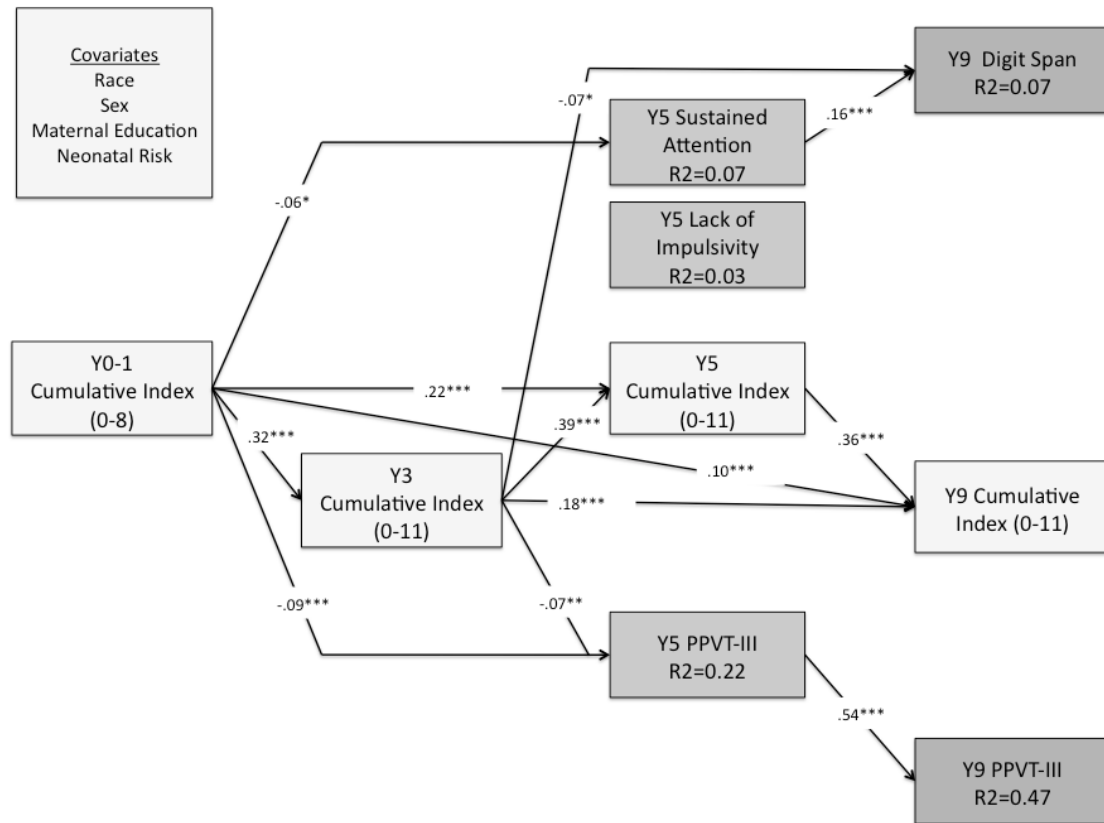
^a Indicators within the same wave were correlated (not depicted in the diagram). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

The control variables explained the greatest amount of variance for all of the cognitive outcomes (6.5% of variance for age five sustained attention, 2.4% of variance for lack of impulsivity, 21.1% of variance for age five PPVT-III, 6.4% of variance for age nine digit span, and 46.3% of variance for age nine PPVT-III). Most notably, higher maternal education significantly predicted better scores on all cognitive outcomes. Significant effects for race and gender were also observed. Non-Hispanic blacks and Hispanics scored significantly lower on the PPVT-III at ages five and nine compared to non-Hispanic whites. Non-Hispanic blacks scored significantly lower than whites on the sustained attention measure at age five, and Hispanics scored significantly lower than

whites on the digit span measure at age nine. Females scored significantly higher than males on the age five sustained attention and lack of impulsivity measures. Addition of the infancy and age three cumulative indices increased the percentage of variance explained for all cognitive outcomes by a negligible amount ($<1\%$), and there was no increase after the addition of the age five and age nine cumulative indices.

Given the significance of the direct paths to and from endogenous variables (specifically, age three cumulative index, age five sustained attention, and age five PPVT-III), we also examined the total and specific indirect effects from the cumulative indices during infancy and at age three to each cognitive outcome. No specific hypotheses were made. Results are shown in Table 4.3. Significant, specific indirect effects were observed between the cumulative adversity index in infancy and all outcomes except for lack of impulsivity. Specific indirect paths from the cumulative adversity index in infancy to: age nine digit span went through the cumulative adversity index at age three ($\beta=-0.02$; $p<0.05$); age five PPVT went through age three cumulative adversity index ($\beta=-0.02$; $p<0.01$); and age nine PPVT went through the age five PPVT ($\beta=-0.05$; $p<0.001$), and through the age three cumulative adversity index and age five PPVT ($\beta=-0.01$; $p<0.01$). Additionally, the specific, indirect effect for the cumulative index at age three was significant for age nine PPVT through age five PPVT ($\beta=-0.04$; $p<0.01$).

Figure 4.3. Hypothesis 2.1b Final Model^a



(*p<0.05; **p<0.01; ***p<0.001; RMSEA=0.06; CFI=0.987)

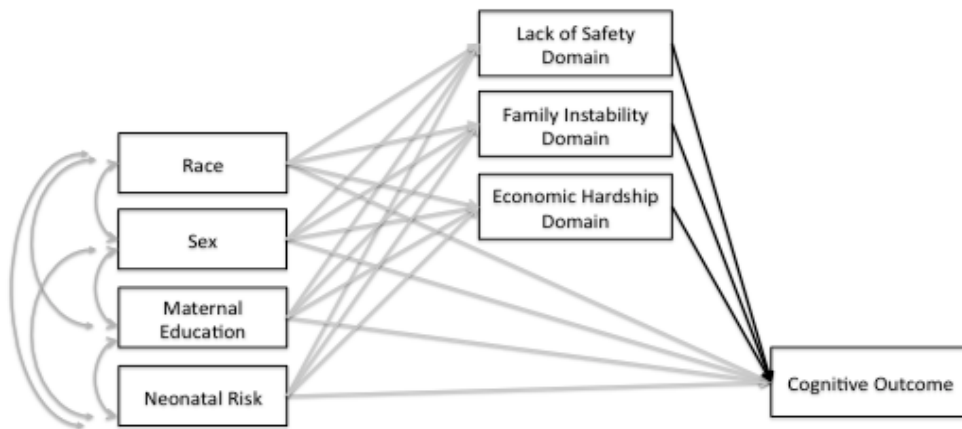
^aStandardized coefficients presented. Indicators within the same wave were correlated in the expected direction (not depicted in the diagram). Cognitive outcomes within the same wave were significantly positively correlated. The age five CI was significantly correlated with age five sustained attention ($r=-0.06^*$) and PPVT-III ($r=-0.08^{**}$). The age nine CI was significantly correlated with age nine digit span ($r=-0.04^*$). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

Relation Between Adversity Domains and Cognitive Outcomes

To test the hypothesis (2.2a) that *higher domain scores at each age would predict worse cognitive scores*, simple path models were examined in which each cognitive outcome was individually regressed on the three adversity domains for each age, one wave at a time, controlling for covariates. In these models, covariates pointed to all of the domains and the cognitive outcomes (shown in Figure 4.4). The significance of the direct path between each domain and each cognitive outcome was used to test the stated

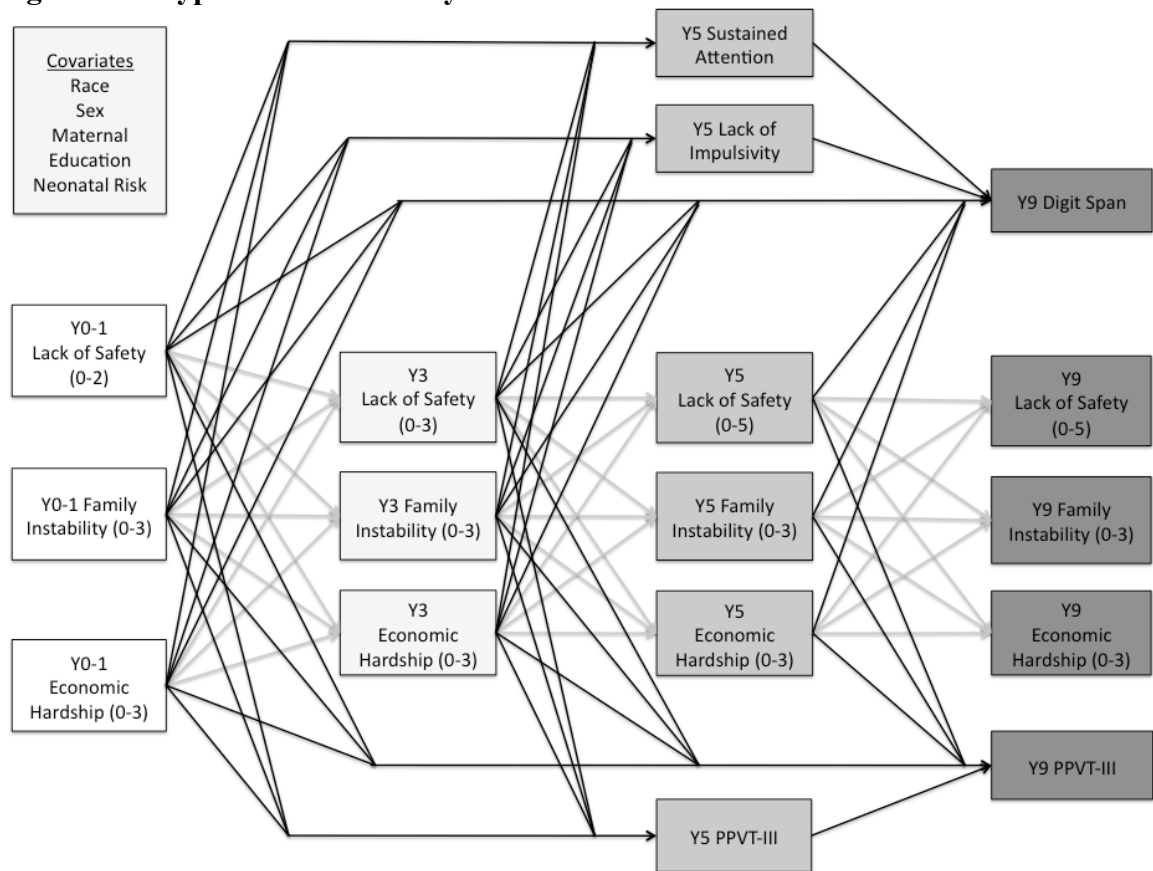
hypothesis (results shown in the Appendix, Table A4.4). All models were a good fit (RMSEA < 0.05; CFI > 0.95). Only the economic hardship domain significantly predicted the cognitive outcomes after controlling for covariates and the other domains within the same wave. Higher scores on the economic hardship domain predicted worse cognitive outcomes. This was true for all waves of adversity domains and for all cognitive outcomes, with the exception that there were no significant effects of any domain at infancy and age three for the lack of impulsivity outcome.

Figure 4.4 Hypothesis 2.2a Analytic Model



The analytic model shown in Figure 4.5 below was used to test the next hypothesis that *higher adversity domain scores during infancy and at age three would directly predict worse cognitive scores at ages five and nine, even after controlling for adverse exposures at ages five and nine*. Again, the model was carefully constructed to watch for any change in variance of the cognitive outcomes with the addition of each wave of adversity domain (model building results are shown in the Appendix, Table A4.6).

Figure 4.5. Hypothesis 2.2b Analytic Model ^a



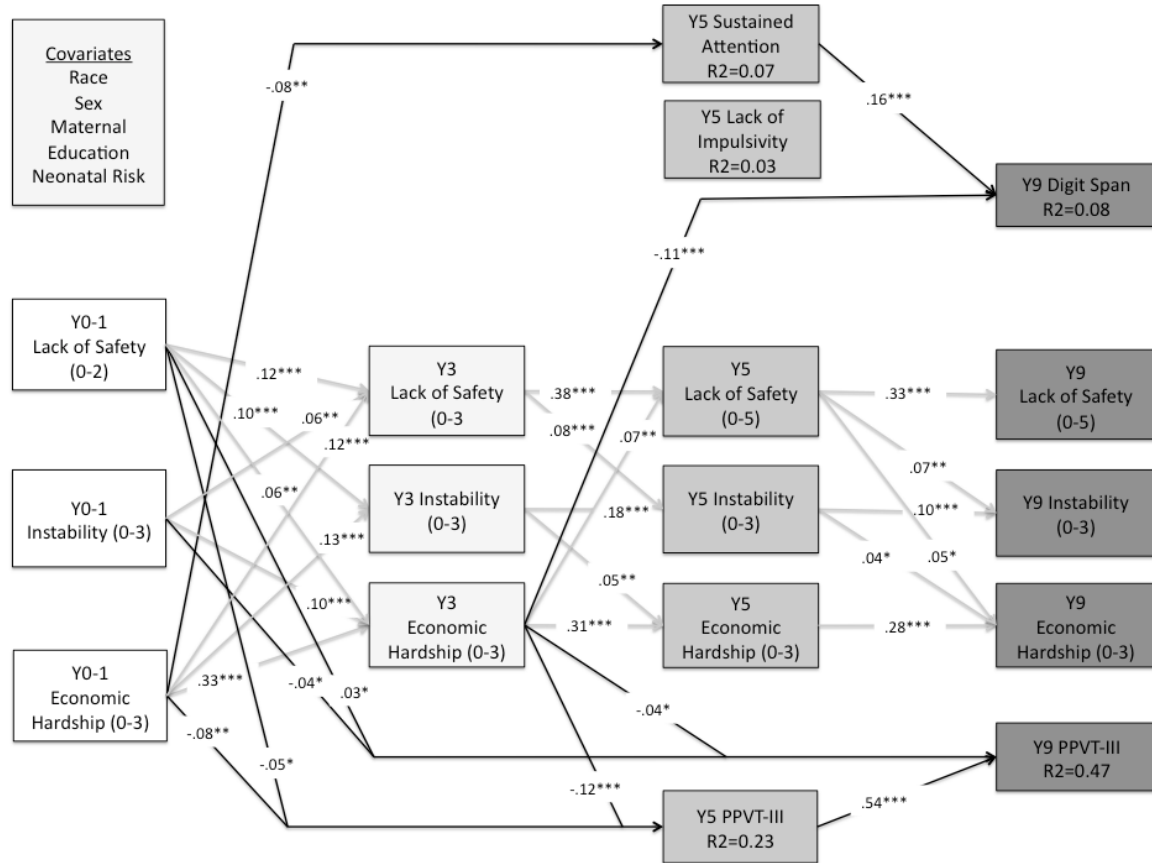
^aThe conceptual model allowed for direct longitudinal paths between all adversity domains (paths representing one lag shown with the grey arrows; paths greater than one lag are not shown). All indicators within the same wave were correlated (not depicted in the diagram). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

The final model is shown in Figure 4.6. After controlling for covariates and all adversity domains at each wave, significant direct effects were observed between adversity domains at infancy and at age three with the cognitive outcomes. Specifically, during infancy, the lack of safety domain directly predicted PPVT-III at age five ($\beta = -0.05$; $p < 0.05$) and PPVT-III at age nine ($\beta = 0.03$; $p < 0.05$), unexpected result; instability directly predicted age nine PPVT ($\beta = -0.04$; $p < 0.05$); and economic hardship predicted age five sustained attention ($\beta = -0.08$; $p < 0.01$) and age five PPVT-III ($\beta = -0.08$; $p < 0.01$). At age three, economic hardship also directly predicted age five PPVT-III ($\beta = -0.12$;

$p < 0.001$), age nine PPVT-III ($\beta = -0.04$; $p < 0.05$), and age nine digit span ($\beta = -0.11$; $p < 0.001$). The addition of the adversity domains at infancy and at age three explained a minimal increase in variance for each of these outcomes (0.1-1.0%) above that explained by the control variables. There was no change in variance explained with the addition of the age five and age nine adversity domains.

Per the nature of longitudinal research, we further explored the specific indirect effects from the infancy and age three domains in relation to the cognitive outcomes, given the significant direct paths to and from endogenous variables (specifically, age three economic hardship domain, age five sustained attention and age five PPVT-III). No specific hypotheses were made. There were significant, negative indirect effects between each adversity domain in infancy and the age five PPVT-III, age nine PPVT-III, and age nine digit span – all mediated by the economic hardship domain at age three (shown in Table 4.4). There were no significant indirect effects of the adversity domains during infancy or age three on the age five sustained attention and lack of impulsivity outcomes.

Figure 4.6. Hypothesis 2.2b Final Model ^a



(*p<0.05; **p<0.01; ***p<0.001; RMSEA=0.04; CFI=0.99)

^a Standardized coefficients presented. Indicators within the same wave were correlated in the expected direction (not depicted in the diagram). Cognitive outcomes within the same wave were significantly positively correlated. At age five, the lack of safety and economic hardship domains were negatively correlated with sustained attention ($r=-0.06^*$ for both), and economic hardship was negatively correlated with the PPVT-III ($r=-0.12^{***}$). At age nine, only the economic hardship domain was negatively correlated with the PPVT-III ($r=-0.06^*$). Direct, significant paths between domains with greater than one lag are also not shown; while these paths were all in the expected direction, not all were significant. The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

DISCUSSION

The current study examined longitudinal relations between multiple adverse exposures and child cognitive outcomes among a relatively disadvantaged sample of urban children. We used two different approaches for studying multiple adversities. The first was a commonly used cumulative adversity index that summed all adverse exposures

into one single score. The second approach used the same measures of adverse experiences, but grouped them into three adversity domains (lack of safety, family instability and economic hardship) to determine whether certain types of adverse exposures differentially impacted cognitive outcomes. As expected, the cumulative adversity measure was negatively associated with sustained attention, working memory/attention (digit span), and verbal ability (PPVT-III). Although we expected that each of our three adversity domains would also predict worse cognitive outcomes, we found the most salient effects for only the economic hardship domain. Confirming our hypothesis regarding the influential effect of early experiences, adverse exposures at infancy and age three directly predicted sustained attention and verbal ability at age five, and working memory/attention, and verbal ability at age nine, even after controlling for concurrent adverse exposures.

Several interesting findings from this study are worthy of discussion. First, the total number of adversities that young people in this study experienced was only moderately stable across early and middle childhood. The variability in total number of adversities and the adversity domain scores increased with time between measurements. Others have shown greater stability in cumulative indices measured over time which (8,17). However, these studies included more stable risk factors/exposures as part of their cumulative indices (i.e., mother's education and race) whereas the current controlled for these more stable factors and focused on adversities that may be more likely to vary over time. In the current study, adverse exposures in the economic hardship domain showed the least variability over time; however, there was still heterogeneity within subjects. Overall, this variability in exposure across time permits a more thorough examination of

how developmental periods during which children experience adversity influences outcomes.

Second, the salient effects of the economic hardship domain in relation to the lack of safety and family instability domains were particularly noteworthy. We expected all three of these adversity domains to predict children's cognitive outcomes. However, in individual models that separately regressed each cognitive outcome on all three adversity domains from the same wave, we found that *only* the economic hardship domain for each wave significantly predicted all five of the cognitive outcomes, controlling for covariates. Additionally, our final model that accounted for all adversity domains from all waves of the study showed that the economic hardship domain remained a significant predictor of sustained attention, working memory/attention, and verbal ability, and the magnitudes of the path coefficients from the economic hardship domains to the cognitive outcomes were comparable to those of the full cumulative adversity indices. These results suggest that economic hardship drives the effect of the cumulative index for these cognitive outcomes.

While the impact of economic hardship, and poverty in particular, on child cognitive development is well documented (5,30,31), the lack of robust effects for the lack of safety and family instability domains were surprising. Despite the biologically plausible mechanism for the lack of safety and family instability domains to influence cognitive outcomes through demands on a child's stress response systems (34,70,71), we did not see strong effects of these domains after accounting for economic hardship. Others who have compared the effect of multiple adversity domains on child cognitive outcomes corroborate these results. Klebanov and Brooks-Gunn (2006) showed only significant effects of a human capital domain, but not a psychological domain on

children's IQ at ages three, five and eight (19), and Schoon et al (2012) showed that economic hardship, but not family instability was significantly associated with cognitive functioning in five year olds (32). Although the effects of economic hardship were most striking, we did observe significant direct effects of the lack of safety and family instability domains at infancy on verbal ability, and indirect effects of these domains on working memory/attention at age nine, mediated by economic hardship at age three. The positive path between family instability at infancy and verbal ability at age nine was unexpected and may be spurious artifact of the model. The total effect of this domain on verbal ability at age nine was not significant, and therefore, we do not give too much weight to this result.

There are several factors that may explain the dominant effects of economic hardship observed in this sample. Our cut-off for our measure of poverty was at or below the federal poverty level. This is an extreme level of poverty and a persistent exposure for children in this sample (approximately 75% of the children living in poverty at one wave were also living in poverty at the subsequent wave). Furthermore, adverse exposures in the lack of safety and family instability domains are more common in conditions of poverty (10,72). Therefore, poverty may have masked the effects of these other, less severe exposures. Additionally, economic hardship may be more likely to influence cognitive assessments whereas unsafe and unstable environments may be more likely to influence social-emotional or behavioral outcomes (19). Further research is needed to examine the effects of these domains on other social, emotional and behavioral outcomes. The selection hypothesis is an alternative explanation for the strong effects of poverty on child cognitive development (73). This hypothesis proposes that parental

characteristics, such as genetics, confound the relationship between poverty and child outcomes. In other words, parents with lower IQs may be more likely to live in poverty, passing along the genetic traits of low IQ to their children. However, the relationship between poverty and cognitive outcomes persisted in this study, even after controlling for maternal education and cognitive ability, suggesting that other factors associated with poverty are at play.

Our third significant finding worth highlighting was that the magnitudes of the significant associations between the adversity measures and cognitive outcomes were modest after accounting for covariates, though comparable to other studies (25,74). For example, the largest coefficients translate to decrements of a quarter of a point for the attention measures or one full point for verbal ability with an increase in the number of adversities. In fact, the control variables (e.g., maternal education, race, and gender) explained most of the variance of the cognitive outcomes; accounting for adverse exposures at each wave only increased the total variance explained by at most two percent. Most notably, children whose mothers had a high school education or less scored significantly lower on all cognitive outcomes. The association of maternal education with child cognitive outcomes and achievement is well documented; more educated mothers tend to exhibit parenting behaviors that foster cognitive development, including more cognitively stimulating language use and activities, more educational resources in the home, and more involvement with the child's schooling (75,76). Additionally, more educated mothers are more likely to delay childbearing and have more educated husbands and higher family income (76). Race was also a significant predictor for all cognitive outcomes in this study with black children scoring lower than

children on sustained attention, lack of impulsivity and verbal ability, and Hispanic children scoring lower than white children on verbal ability and working memory/attention. Worth noting, a factor such as race does not represent biological risk per se but rather a constellation of exposures, such as discrimination or cultural differences associated with race that are otherwise unmeasured (77).

Our fourth finding pertains to the lasting effect of adverse exposures experienced by age three on cognitive outcomes in middle and late childhood. This study was unique in that it assessed multiple adverse exposures at four different ages across childhood and the direct and indirect effects of exposure at each of these time points on cognitive outcomes. Exposures that occurred at the time of the child's birth – including both adverse exposures and the mother's level of education – explained most of the variance in each of these cognitive outcomes. Additionally, after accounting for all exposures from all waves of the study, both the cumulative adversity model and the adversity domain model showed significant direct and indirect effects of adverse exposures at infancy and age three on child sustained attention, working memory/attention and verbal ability during pre-school and late childhood periods; these direct and indirect effects persisted, even after accounting for later adversities that occurred during the time the cognitive outcomes were assessed. The lasting impact of early adverse exposures provides some evidence for an early sensitive period present in the first three years of a child's life. It is important to highlight that this study did not decompose adverse exposures that *only* occurred in this early period, and it is likely that for many children, adversity in early childhood persisted throughout their development. However, adversities experienced by age three predicted later cognitive development, regardless of later exposures. These

findings add to a growing body of evidence for an early sensitive period for cognitive development (44,78).

Finally, we found no significant effects of either the cumulative adversity index or the adversity domains on the lack of impulsivity outcome. Gender was the only significant predictor for this outcome, with females scoring higher than males. While sustained attention and lack of impulsivity are both measures of attention, they represent different attention networks in the brain (79). This may explain the absence of any significant findings for the lack of impulsivity outcome. The sustained attention measure represents the alerting attentional system which is involved in maintaining a vigilant and alert state and primarily responds to the neurotransmitter norepinephrine (80). Lack of impulsivity is a measure of the executive control network involved in self-regulation and responds primarily to the neurotransmitter dopamine (80). A study by Rueda et al (2004) showed differing developmental trajectories of these two attention networks. Among a sample of six to ten year olds, alerting attention improved with age, though children differed substantially from adults suggesting protracted development in this system. Executive attention also improved from six to eight years, and then remained fairly stable and comparable to adults (81). While the current study was unable to determine why adversities would differentially affect these two attention measures, nuanced differences in these two systems suggest different pathways are plausible. This noted, others have shown better performance on both of these measures of attention by more socioeconomically advantaged children (82).

Limitations

There are a few limitations to this study worth noting. First, the measures of adversity used in this study were obtained from the biological mother. Due to socially desirable responses, particularly with respect to topics such as child maltreatment or domestic violence, these data may underestimate the actual occurrence of adversities and their relations to cognitive outcomes. As well, there were no child reports of adversity exposure, so an assumption was made that children experienced the adversities reported by the mother. Second, the Leiter sustained attention and lack of impulsivity measures assessed at age five measure different aspects of attention from the WISC-IV digit span assessed at age nine, limiting our ability to understand longitudinal changes in these constructs. Moreover, each construct is assessed with only one instrument. Third, we hypothesized that adverse experiences would influence measures of attention via their impact on the stress response system. However, we had no measures of stress in this study. Further research would benefit by examining the relation between adversity domains and biomarkers of stress reactivity. Finally, while the longitudinal nature of these data is strength of the study, the timing of these assessments also presents a study limitation. The exact ages of children varied for each wave. Therefore, the study conclusions about temporality can only be generalized to early, middle, and late childhood rather than specific ages.

Implications and Conclusion

This study has several important implications. First, a domain-based approach to studying the effects of multiple adverse experiences is more informative for identifying

intervention targets. While it is easy to argue that all adversity is detrimental to a child's development, limited resources dictate prioritizing intervention efforts. This study indicates that policies and programs that focus on alleviating economic hardship and bolstering the education of mothers or future mothers should be prioritized in order to improve children's cognitive development. A positive example is the Earned Income Tax Credit (EITC), which directly supplements the income of low-income workers. One of the many benefits of the EITC is improved cognitive performance for children of recipients (83). Additionally, efforts to educate girls may have intergenerational effects on their children's cognitive development. Increasing maternal education during the early years of their child's life has even been shown to improve cognitive outcomes for children (75).

There has been increased focus by pediatricians and public health practitioners to screen children for adverse experiences and to connect children experiencing high levels of adversity to appropriate services (10,84,85). Many of these screening tools do not currently assess for economic hardship; however, results from this study indicate that economic hardship should be included in any screening tool. This study also has implications for intervention timing and lends support for programs such as home visiting (86) and early childhood education (87) that intervene within the first few years of a child's life. These programs aim to bolster development of key social, emotional, cognitive and health outcomes and have been shown to reduce disparities in achievement evident by the time that socioeconomically disadvantaged children enter kindergarten (86,87).

In conclusion, this study shows that adverse exposures in the first three years of life have lasting effects, though modest, on assessments of sustained attention, working memory/attention and verbal ability in middle and late childhood. Furthermore, economic hardship accounts for the effect of cumulative adversity on these cognitive outcomes. Future research should examine the effect of adversity domains on different social, emotional and behavioral outcomes. Policy and programs that reduce poverty and bolster maternal education in early childhood are warranted.

Table 4.1. Description of Key Variables (N=2976)

Final Analytic Sample				
	N	%	% Missing	
Mothers with \leq High School Education at Child's Birth	1087	36.6	0.1	
Mother Race			0.2	
<i>White, non-Hispanic</i>	591	19.9		
<i>Black, non-Hispanic</i>	1494	50.3		
<i>Hispanic</i>	784	26.4		
<i>Other</i>	100	3.4		
Female Child	1428	48.0	0	
Neonatal Risk	317	10.7	0.7	
	Mean	SD	% Missing	Range
Mean Maternal Age	25.1	6.0	<0.1	15-43
Year 5 Cognitive Outcomes ^a				
<i>Sustained Attention</i>	12.9	3.3	44.3	1-19
<i>Impulse Control</i>	10.1	2.9	44.3	1-17
<i>PPVT</i>	94.3	15.4	38.1	40-139
Year 9 Cognitive Outcomes ^a				
<i>Digit Span</i>	9.4	2.8	0.2	1-19
<i>PPVT</i>	93.1	14.9	0.5	53-159
Year 1 Cumulative Index	1.4	1.2	11.4	0-8
Year 3 Cumulative Index	2.1	1.7	30.2	0-10
Year 5 Cumulative Index	2.2	1.8	34.6	0-9
Year 9 Cumulative Index	2.3	1.7	16.1	0-9
Year 1 Lack of Safety	0.2	0.4	7.3	0-2
Year 1 Instability	0.6	0.7	9.8	0-3
Year 1 Economic Hardship	0.6	0.7	5.8	0-3
Year 3 Lack of Safety	1.1	1.1	25.1	0-5
Year 3 Instability	0.4	0.6	11.5	0-3
Year 3 Economic Hardship	0.6	0.7	25.1	0-3
Year 5 Lack of Safety	1.2	1.1	27.8	0-5
Year 5 Instability	0.3	0.5	15.5	0-3
Year 5 Economic Hardship	0.7	0.8	5.9	0-3
Year 9 Lack of Safety	1.4	1.2	9.2	0-5
Year 9 Instability	0.3	1.1	7.9	0-3
Year 9 Economic Hardship	0.7	0.8	1.5	0-3

^a Standardized scores presented here.

Table 4.2. Correlations Among All Variables ^a

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)	(21)	(22)	(23)	(24)	(25)	(26)	(27)	(28)
(1) Y9 Digit Span	-																											
(2) Y5 Sust. Attn.	.17	-																										
(3) Y5 Lack of Imp.	.08	.15	-																									
(4) Y9 PPVT	.34	.24	.13	-																								
(5) Y5 PPVT	.25	.34	.16	.65	-																							
(6) White	.09	.08	.06	.35	.35	-																						
(7) Black	-.01	-.18	-.11	-.29	-.20	-	-																					
(8) Hispanic	-.12	.11	.05	-.13	-.22	-	-	-																				
(9) Other	.08	.07	.09	.22	.19	-	-	-	-																			
(10) Sex	.03	.22	.14	-.04	.11	-	-	-	-	-																		
(11) > HS Edu.	.20	.16	.11	.45	.44	.28	-.14	-.25	.19	.00	-																	
(12) Neonatal Risk	-.08	-.12	-.03	-.12	-.08	-.08	.26	-.21	-.17	.05	-.05	-																
(13) Y1 CI	-.09	-.11	-.06	-.26	-.24	-.24	.33	-.05	-.23	-.01	-.41	.10	-															
(14) Y3 CI	-.12	-.09	-.06	-.27	-.23	-.26	.37	-.11	-.07	-.05	-.40	.10	.44	-														
(15) Y5 CI	-.07	-.12	-.09	-.25	-.24	-.25	.36	-.09	-.10	-.04	-.35	.08	.44	.54	-													
(16) Y9 CI	-.11	-.07	-.09	-.20	-.13	-.19	.28	-.09	-.09	-.04	-.31	.07	.37	.46	.54	-												
(17) Y1 Unsafe	-.05	-.05	-.04	-.10	-.13	-.10	.09	.04	-.17	.00	-.22	.03	.54	.24	.23	.20	-											
(18) Y1 Instability	-.04	-.05	-.04	-.18	-.11	-.20	.36	-.18	-.16	-.01	-.23	.07	.72	.26	.31	.24	.12	-										
(19) Y1 Hardship	-.11	-.12	-.07	-.24	-.24	-.18	.17	.05	-.13	-.01	-.38	.08	.77	.39	.36	.32	.23	.26	-									
(20) Y3 Unsafe	-.05	-.04	-.06	-.17	-.16	-.19	.31	-.15	-.03	-.09	-.26	.05	.28	.80	.45	.39	.18	.18	.22	-								
(21) Y3 Instability	-.03	-.03	-.04	-.08	-.06	-.08	.17	-.09	-.09	.01	-.14	.04	.20	.58	.23	.21	.14	.10	.18	.20	-							
(22) Y3 Hardship	-.17	-.11	-.08	-.29	-.26	-.22	.21	.04	-.10	-.01	-.43	.11	.45	.66	.44	.33	.19	.25	.44	.26	.38	-						
(23) Y5 Unsafe	-.04	-.10	-.04	-.16	-.15	-.22	.31	-.11	-.06	-.06	-.22	.05	.29	.43	.82	.45	.19	.21	.21	.46	.22	.25	-					
(24) Y5 Instability	-.03	-.01	-.06	-.09	-.07	-.08	.15	-.06	-.15	-.01	-.09	.01	.19	.24	.53	.24	.05	.19	.14	.16	.11	.13	.20	-				
(25) Y5 Hardship	-.09	-.12	-.09	-.25	-.26	-.20	.22	-.14	-.04	.01	-.35	.10	.41	.41	.68	.38	.18	.25	.29	.21	.22	.48	.24	.29	-			
(26) Y9 Unsafe	-.07	-.04	-.07	-.11	-.10	-.17	.23	-.06	-.04	-.07	-.16	.02	.24	.35	.43	.82	.15	.15	.19	.38	.19	.17	.45	.23	.20	-		
(27) Y9 Instability	-.02	-.02	-.04	-.07	-.01	-.04	.21	-.07	-.06	.01	-.16	.05	.20	.19	.19	.49	.12	.12	.13	.11	.11	.12	.14	.15	.14	.15	-	
(28) Y9 Hardship	-.10	-.08	-.07	-.22	-.16	-.16	.20	-.02	-.17	.01	-.34	.07	.34	.38	.41	.68	.14	.14	.33	.22	.18	.40	.22	.18	.45	.24	.21	-

^a Z-scores used for cognitive outcomes. Sust. Attn. = sustained attention; Lack of Imp = lack of impulsivity; HS edu = high school education; CI=cumulative index; Y=year

Table 4.3. Total, Direct and Specific Indirect Effects of Cumulative Adversity Indices on Cognitive Outcomes^a

Path Model	Total Effect	Direct Effect	Specific Indirect Effects
	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)
Y1 CI → Y5 Sustained Attention	-0.054 (0.026)*	-0.055 (0.028)*	
Y1 CI → Y5 Lack of Impulsivity	-0.033 (0.027)	-0.031 (0.029)	
Y1 CI → Y5 PPVT	-0.114 (0.022)***	-0.090 (0.023)***	
Y1CI → Y3CI → Y5PPVT			-0.024 (0.008)**
Y1 CI → Y9 Digit Span	-0.047 (0.021)*	-0.020 (0.023)	
Y1CI → Y3CI → Y9DS			-0.02 (0.009)*
Y1 CI → Y9 PPVT	-0.096 (0.018)***	-0.023 (0.019)	
Y1CI → Y3CI → Y5PPVT → Y9PPVT			-0.013 (0.004)**
Y1CI → Y5PPVT → Y9PPVT			-0.049 (0.013)***
Y3 CI → Y9 Digit Span	-0.063 (0.025)*	-0.040 (0.016)*	
Y3 CI → Y9 PPVT	-0.073 (0.022)***	-0.031 (0.022)	
Y3CI → Y5PPVT → Y9PPVT			-0.040 (0.013)**

*p<0.05; **p<0.01; ***p<0.001; CI=cumulative index; Y1=year 1; Y3=year 3; Y5=year 5; Y9=year 9; PPVT=Peabody Picture Vocabulary-III; DS=digit span

^aTotal effect refers to the sum of all paths from the exogenous to the endogenous variable. Direct effect refers to only the direct path from the exogenous to the endogenous variable. Specific indirect effect refers to the path from the exogenous to the endogenous variable through a specific intermediary variable.

Table 4.4. Total, Direct and Specific Indirect Effects of Domains on Cognitive Outcomes

	Total Effect Standardized β (SE)	Direct Effect Standardized β (SE)	Specific Ind. Effect Standardized β (SE)
<i>Lack of Safety Domain</i>			
Y1LS \rightarrow Y5 Sustained Attention	-0.020 (0.025)	-0.025 (0.025)	
Y1LS \rightarrow Y5 Lack of Impulsivity	-0.018 (0.026)	-0.013 (0.026)	
Y1LS \rightarrow Y5 PPVT	-0.058 (0.020)**	-0.052 (0.021)*	
<i>Y1LS \rightarrow Y3EH \rightarrow Y5PPVT</i>			-0.007 (0.003)*
Y1LS \rightarrow Y9 Digit Span	-0.013 (0.019)	-0.007 (0.019)	
<i>Y1LS \rightarrow Y3EH \rightarrow Y9DS</i>			-0.007 (0.003)*
Y1LS \rightarrow Y9 PPVT	-0.002 (0.017)	0.033 (0.016)*	
<i>Y1LS \rightarrow Y5PPVT \rightarrow Y9PPVT</i>			-0.028 (0.011)*
<i>Y1LS \rightarrow Y3EH \rightarrow Y5PPVT \rightarrow Y9PPVT</i>			-0.004 (0.001)*
Y3LS \rightarrow Y9 Digit Span	0.013 (0.023)	0.002 (0.025)	
Y3LS \rightarrow Y9 PPVT	-0.022 (0.020)	-0.015 (0.020)	
<i>Family Instability Domain</i>			
Y1FI \rightarrow Y5 Sustained Attention	-0.028 (0.026)	-0.028 (0.026)	
Y1FI \rightarrow Y5 Lack of Impulsivity	-0.003 (0.027)	-0.006 (0.027)	
Y1FI \rightarrow Y5 PPVT	0.015 (0.022)	0.027 (0.022)	
<i>Y1FI \rightarrow Y3EH \rightarrow Y5PPVT</i>			-0.011 (0.003)***
Y1FI \rightarrow Y9 Digit Span	0.005 (0.020)	0.009 (0.021)	
<i>Y1FI \rightarrow Y3EH \rightarrow Y9DS</i>			-0.011 (0.003)***
Y1FI \rightarrow Y9 PPVT	-0.033 (0.018)	-0.037 (0.017)*	
<i>Y1FI \rightarrow Y3EH \rightarrow Y5PPVT \rightarrow Y9PPVT</i>			-0.006 (0.002)***
Y3FI \rightarrow Y9 Digit Span	0.010 (0.020)	0.010 (0.020)	
Y3FI \rightarrow Y9 PPVT	0.021 (0.018)	0.009 (0.017)	
<i>Economic Hardship Domain</i>			
Y1EH \rightarrow Y5 Sustained Attention	-0.084 (0.026)***	-0.079 (0.028)**	
Y1EH \rightarrow Y5 Lack of Impulsivity	-0.041 (0.027)	-0.031 (0.029)	
Y1EH \rightarrow Y5 PPVT	-0.116 (0.022)***	-0.080 (0.023)***	
<i>Y1EH \rightarrow Y3EH \rightarrow Y5PPVT</i>			-0.037 (0.008)***
Y1EH \rightarrow Y9 Digit Span	-0.056 (0.020)**	-0.012 (0.022)	
<i>Y1EH \rightarrow Y5SA \rightarrow Y9DS</i>			-0.013 (0.005)*
<i>Y1EH \rightarrow Y3EH \rightarrow Y9DS</i>			-0.037 (0.009)***
Y1EH \rightarrow Y9 PPVT	-0.096 (0.013)***	-0.019 (0.018)	
<i>Y1EH \rightarrow Y5PPVT \rightarrow Y9PPVT</i>			-0.043 (0.013)***
<i>Y1EH \rightarrow Y3EH \rightarrow Y9PPVT</i>			-0.013 (0.007)*
<i>Y1EH \rightarrow Y3EH \rightarrow Y5PPVT \rightarrow Y9PPVT</i>			-0.020 (0.005)***
Y3EH \rightarrow Y9 Digit Span	-0.113 (0.024)***	-0.114 (0.026)***	
Y3EH \rightarrow Y9 PPVT	-0.103 (0.022)***	-0.041 (0.021)*	
<i>YEH \rightarrow Y5PPVT \rightarrow Y9PPVT</i>			-0.062 (0.014)*

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Y1=year 1; Y3=year 3; Y5=year 5; Y9=year 9; PPVT=Peabody Picture Vocabulary-III; DS=digit span; EH=economic hardship ; LS=lack of safety ; FI=family instability

APPENDIX, Table A4.1. Bivariate Relations Between Individual Adversities and Cognitive Outcomes ^a

	Y5 Sustained Attention	Y5 Lack of Impulsivity	Y5 PPVT-III	Y9 Digit Span	Y9 PPVT-III
	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)
<i>Covariates</i>					
White (ref)	---	---	---	---	---
Other	-0.003 (0.026)	0.014 (0.026)	-0.055 (0.023)*	0.000 (0.019)	-0.024 (0.018)
Black	-0.148 (0.031)***	-0.094 (0.032)**	-0.423 (0.027)***	-0.096 (0.024)***	-0.461 (0.021)***
Hispanic	-0.007 (0.031)	-0.021 (0.031)	-0.418 (0.027)***	-0.145 (0.024)***	-0.374 (0.022)***
Female	0.179 (0.024)***	0.108 (0.024)***	0.086 (0.023)***	0.027 (0.018)	-0.032 (0.018)
< H.S. Ed.	-0.119 (0.024)***	-0.087 (0.024)***	-0.337 (0.021)***	-0.161 (0.018)***	-0.367 (0.016)***
Neonatal Risk	-0.072 (0.025)**	-0.018 (0.025)	-0.049 (0.023)*	-0.050 (0.018)**	-0.070 (0.018)***
<i>Y1 Exposures</i>					
Maternal Depression	-0.010 (0.025)	-0.011 (0.025)	-0.010 (0.024)	-0.006 (0.019)	-0.029 (0.019)
IPV	0.014 (0.025)	0.017 (0.025)	-0.031 (0.024)	-0.021 (0.019)	-0.016 (0.019)
Single Parent	-0.050 (0.025)*	-0.039 (0.025)	-0.115 (0.023)***	-0.057 (0.018)**	-0.318 (0.037)***
Father Incarceration	-0.028 (0.025)	-0.011 (0.025)	-0.046 (0.024)	0.017 (0.019)	-0.108 (0.019)***
Unsafe Neighborhood	-0.068 (0.024)**	-0.059 (0.025)*	-0.140 (0.023)***	-0.047 (0.018)*	-0.113 (0.018)***
Below Poverty Level	-0.123 (0.024)***	-0.086 (0.024)***	-0.273 (0.022)***	-0.108 (0.018)***	-0.286 (0.017)***
Housing Insecurity	-0.077 (0.015)**	-0.026 (0.025)	-0.109 (0.023)***	-0.046 (0.019)*	-0.072 (0.019)***
Food Insecurity	-0.008 (0.025)	0.017 (0.025)	-0.009 (0.024)	-0.008 (0.019)	-0.013 (0.019)
<i>Y3 Exposures</i>					
Psych. Aggression	-0.018 (0.027)	-0.016 (0.027)	-0.058 (0.025)*	-0.020 (0.021)	-0.057 (0.021)**
Corporal Punishment	0.010 (0.027)	-0.093 (0.027)***	-0.090 (0.025)***	-0.029 (0.021)	-0.071 (0.021)***
Neglect	0.023 (0.027)	0.022 (0.027)	-0.088 (0.025)***	-0.054 (0.021)**	-0.074 (0.021)***
Maternal Depression	0.006 (0.025)	-0.041 (0.025)	-0.004 (0.024)	-0.023 (0.019)	-0.015 (0.019)
IPV	-0.013 (0.025)	0.011 (0.025)	-0.032 (0.024)	0.010 (0.019)	-0.022 (0.019)
Relationship Stability	-0.025 (0.025)	-0.036 (0.025)	-0.041 (0.024)	-0.014 (0.019)	-0.040 (0.019)*
Father Incarceration	-0.033 (0.025)	-0.002 (0.025)	-0.053 (0.024)*	-0.005 (0.019)	-0.083 (0.019)***
Community Violence	-0.078 (0.026)**	-0.061 (0.027)*	-0.152 (0.015)***	-0.027 (0.021)	-0.208 (0.020)***
Below Poverty Level	-0.112 (0.025)***	-0.087 (0.025)***	-0.301 (0.022)***	-0.154 (0.019)***	-0.327 (0.017)***
Housing Insecurity	-0.026 (0.025)	-0.019 (0.025)	-0.058 (0.024)*	-0.063 (0.019)***	-0.065 (0.019)***
Food Insecurity	-0.007 (0.027)	-0.019 (0.027)	-0.083 (0.025)***	-0.067 (0.021)***	-0.077 (0.021)***

APPENDIX, Table A4.1. Bivariate Relations Between Individual Adversities and Cognitive Outcomes (continued) ^a

	Y5 Sustained Attention Standardized β (SE)	Y5 Lack of Impulsivity Standardized β (SE)	Y5 PPVT-III Standardized β (SE)	Y9 Digit Span Standardized β (SE)	Y9 PPVT-III Standardized β (SE)
<i>Y5 Exposures</i>					
Psych. Aggression	-0.072 (0.025)**	-0.032 (0.025)	-0.065 (0.023)**	0.000 (0.021)	-0.061 (0.021)**
Corporal Punishment	-0.049 (0.025)*	-0.003 (0.025)	-0.090 (0.025)***	-0.023 (0.021)	-0.098 (0.021)***
Neglect	-0.031 (0.025)	0.012 (0.025)	-0.050 (0.023)*	-0.007 (0.027)	-0.015 (0.021)
Maternal Depression	-0.005 (0.025)	-0.035 (0.025)	-0.033 (0.023)	-0.031 (0.019)	-0.018 (0.019)
IPV	-0.054 (0.025)*	-0.016 (0.025)	-0.056 (0.023)*	-0.008 (0.019)	-0.049 (0.019)*
Relationship Stability	-0.012 (0.025)	-0.037 (0.025)	-0.174 (0.077)*	-0.019 (0.019)	-0.056 (0.019)**
Father Incarceration	-0.020 (0.025)	-0.048 (0.025)	-0.064 (0.024)**	0.005 (0.019)	-0.082 (0.019)***
Community Violence	-0.077 (0.025)**	-0.041 (0.025)	-0.144 (0.023)***	-0.051 (0.021)*	-0.170 (0.021)***
Below Poverty Level	-0.129 (0.024)***	-0.087 (0.024)***	-0.322 (0.021)***	-0.103 (0.019)***	-0.326 (0.017)***
Housing Insecurity	-0.042 (0.025)	-0.044 (0.025)	-0.090 (0.023)***	-0.046 (0.019)*	-0.062 (0.019)***
Food Insecurity	-0.052 (0.025)*	-0.038 (0.025)	-0.066 (0.023)**	0.001 (0.019)	-0.045 (0.019)*
<i>Y9 Exposures</i>					
Psych. Aggression				-0.032 (0.019)	-0.015 (0.019)
Corporal Punishment				-0.093 (0.038)*	-0.066 (0.019)***
Neglect				-0.012 (0.019)	0.012 (0.019)
Maternal Depression				-0.002 (0.019)	-0.023 (0.019)
IPV				-0.024 (0.018)	-0.020 (0.018)
Relationship Stability				-0.025 (0.019)	-0.024 (0.019)
Father Incarceration				0.000 (0.018)	-0.063 (0.018)***
Community Violence				-0.052 (0.018)**	-0.198 (0.018)***
Below Poverty Level				-0.130 (0.018)***	-0.287 (0.017)***
Housing Insecurity				-0.035 (0.018)	-0.056 (0.018)**
Food Insecurity				-0.012 (0.018)	-0.049 (0.018)**

*p<0.05; **p<0.01; ***p<0.001; H.S. Ed=high school education; Psych=psychological; IPV=intimate partner violence.

^a Each outcome (Z-scores) examined in a separate model. Complete case analysis.

APPENDIX, Table A4.2. Adjusted Relations Between Individual Adversities and Cognitive Outcomes ^a

	Y5 Sustained Attention Standardized β (SE)	Y5 Lack of Impulsivity Standardized β (SE)	Y5 PPVT-III Standardized β (SE)	Y9 Digit Span Standardized β (SE)	Y9 PPVT-III Standardized β (SE)
<i>Covariate Block</i>					
White (ref)	--- ---	--- ---	--- ---	--- ---	--- ---
Other	-0.004 (0.026)	0.016 (0.027)	-0.058 (0.023)*	0.002 (0.019)	-0.019 (0.017)
Black	-0.116 (0.032)***	-0.073 (0.033)*	-0.344 (0.027)***	-0.053 (0.024)*	-0.376 (0.021)***
Hispanic	-0.015 (0.033)	-0.004 (0.034)	-0.356 (0.028)***	-0.104 (0.024)***	-0.283 (0.021)***
Female	0.178 (0.023)***	0.107 (0.024)***	0.085 (0.021)***	0.029 (0.018)	-0.030 (0.016)
< H.S. Ed.	-0.100 (0.025)***	-0.072 (0.025)**	-0.256 (0.021)***	-0.049 (0.018)**	-0.297 (0.016)***
Neonatal Risk	-0.061 (0.024)**	-0.011 (0.024)	-0.036 (0.021)	-0.140 (0.018)***	-0.042 (0.016)*
<i>Y1 Exposures</i>					
Maternal Depression	-0.004 (0.041)	-0.010 (0.039)	-0.000 (0.038)	-0.008 (0.030)	-0.023 (0.027)
Domestic Violence	-0.039 (0.047)	0.046 (0.029)	-0.005 (0.051)	-0.015 (0.037)	0.028 (0.040)
Single Parent	-0.008 (0.033)	-0.006 (0.034)	-0.023 (0.032)	-0.042 (0.025)	-0.060 (0.024)*
Father Incarceration	-0.011 (0.048)	0.021 (0.051)	-0.025 (0.054)	0.072 (0.036)*	-0.093 (0.043)*
Unsafe Neighborhood	-0.057 (0.035)	-0.060 (0.039)	-0.098 (0.035)**	-0.030 (0.029)	-0.060 (0.030)*
Below Poverty Level	-0.110 (0.024)**	-0.077 (0.039)*	-0.181 (0.041)***	-0.077 (0.027)**	-0.203 (0.025)***
Housing Insecurity	-0.076 (0.033)*	-0.017 (0.034)	-0.099 (0.032)**	-0.044 (0.027)	-0.046 (0.025)
Food Insecurity	-0.029 (0.052)	0.051 (0.056)	-0.019 (0.049)	-0.000 (0.044)	-0.009 (0.040)
<i>Y3 Exposures</i>					
Psych. Aggression	-0.030 (0.039)	-0.011 (0.037)	-0.019 (0.040)	-0.010 (0.032)	-0.022 (0.033)
Corporal Punishment	0.085 (0.027)*	-0.082 (0.036)*	-0.034 (0.052)	-0.024 (0.030)	-0.007 (0.041)
Neglect	0.070 (0.027)	0.056 (0.044)	-0.074 (0.058)	-0.072 (0.039)	-0.056 (0.042)
Maternal Depression	-0.015 (0.053)	-0.057 (0.038)	-0.001 (0.037)	-0.035 (0.030)	-0.015 (0.027)
Domestic Violence	-0.015 (0.053)	0.030 (0.048)	-0.046 (0.051)	0.037 (0.041)	-0.018 (0.034)
Relationship Stability	-0.019 (0.037)	-0.041 (0.037)	-0.020 (0.043)	-0.005 (0.032)	-0.001 (0.036)
Father Incarceration	0.008 (0.048)	0.042 (0.046)	0.022 (0.051)	0.031 (0.038)	-0.029 (0.041)
Community Violence	-0.038 (0.038)	-0.039 (0.037)	-0.069 (0.033)*	0.012 (0.030)	-0.131 (0.027)***
Below Poverty Level	-0.094 (0.038)*	-0.078 (0.040)*	-0.209 (0.046)***	-0.146 (0.029)***	-0.248 (0.030)***
Housing Insecurity	-0.010 (0.036)	-0.012 (0.035)	-0.030 (0.037)	-0.075 (0.030)*	-0.032 (0.030)
Food Insecurity	-0.022 (0.065)	-0.018 (0.059)	-0.107 (0.057)	-0.137 (0.050)**	-0.116 (0.046)*

APPENDIX, Table A4.2. Adjusted Relations Between Individual Adversities and Cognitive Outcomes (continued) ^a

	Y5 Sustained Attention Standardized β (SE)	Y5 Lack of Impulsivity Standardized β (SE)	Y5 PPVT-III Standardized β (SE)	Y9 Digit Span Standardized β (SE)	Y9 PPVT-III Standardized β (SE)
<i>Y5 Exposures</i>					
Psych. Aggression	-0.046 (0.032)	-0.012 (0.032)	-0.014 (0.039)	0.021 (0.029)	-0.011 (0.033)
Corporal Punishment	-0.002 (0.033)	-0.037 (0.034)	-0.012 (0.053)	-0.008 (0.031)	-0.023 (0.040)
Neglect	-0.048 (0.041)	0.026 (0.040)	-0.041 (0.042)	-0.004 (0.038)	-0.016 (0.034)
Maternal Depression	0.002 (0.041)	-0.055 (0.041)	-0.060 (0.047)	-0.055 (0.033)	-0.026 (0.035)
Domestic Violence	-0.098 (0.048)*	-0.025 (0.047)	-0.068 (0.045)	0.002 (0.038)	-0.057 (0.033)
Relationship Stability	-0.012 (0.040)	-0.055 (0.043)	-0.046 (0.040)	-0.017 (0.032)	-0.051 (0.031)
Father Incarceration	0.033 (0.046)	-0.045 (0.049)	-0.002 (0.058)	0.050 (0.040)	-0.026 (0.048)
Community Violence	-0.032 (0.035)	-0.007 (0.035)	-0.068 (0.033)*	-0.032 (0.032)	-0.091 (0.030)**
Below Poverty Level	-0.109 (0.036)**	-0.074 (0.037)*	-0.251 (0.043)***	-0.068 (0.029)*	-0.253 (0.026)***
Housing Insecurity	-0.039 (0.035)	-0.050 (0.035)	-0.083 (0.039)*	-0.056 (0.029)*	-0.032 (0.032)
Food Insecurity	-0.071 (0.050)	-0.057 (0.046)	-0.076 (0.057)	0.016 (0.037)	-0.046 (0.047)
<i>Y9 Exposures</i>					
Psych. Aggression				-0.028 (0.023)	-0.014 (0.022)
Corporal Punishment				-0.048 (0.026)	-0.002 (0.041)
Neglect				-0.008 (0.026)	0.035 (0.024)
Maternal Depression				-0.016 (0.033)	0.003 (0.040)
Domestic Violence				-0.031 (0.036)	-0.021 (0.033)
Relationship Stability				-0.038 (0.032)	-0.029 (0.033)
Father Incarceration				0.036 (0.043)	-0.009 (0.043)
Community Violence				-0.037 (0.027)	-0.155 (0.028)***
Below Poverty Level				-0.118 (0.026)***	-0.213 (0.024)***
Housing Insecurity				-0.032 (0.026)	-0.012 (0.026)
Food Insecurity				-0.000 (0.035)	-0.041 (0.034)

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; H.S. Ed=high school education; Psych=psychological; IPV=intimate partner violence. ^a Each outcome (Z-scores) examined in a separate model. All models a close fit according to fit statistics. Models adjusted for race, maternal education, gender and neonatal risk.

APPENDIX, Table A4.3. Bivariate Relations Between Cumulative Adversities, Domains and Cognitive Outcomes^a

	Y5 Sustained Attention	Y5 Lack of Impulsivity	Y5 PPVT-III	Y9 Digit Span	Y9 PPVT-III
	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)
<i>Cumulative Adversities</i>					
Y1 Cumulative Index	-0.109 (0.025)***	-0.065 (0.025)*	-0.244 (0.023)***	-0.093 (0.019)***	-0.194 (0.019)***
Y3 Cumulative Index	-0.088 (0.028)***	-0.062 (0.028)*	-0.231 (0.025)***	-0.120 (0.022)***	-0.268 (0.020)***
Y5 Cumulative Index	-0.123 (0.026)***	-0.087 (0.026)***	-0.129 (0.024)***	-0.071 (0.023)**	-0.245 (0.021)***
Y9 Cumulative Index				-0.111 (0.020)***	-0.194 (0.019)***
<i>Domains</i>					
Y1 Lack of Safety	-0.050 (0.025)*	-0.039 (0.025)	-0.135 (0.023)***	-0.050 (0.019)**	-0.103 (0.019)***
Y1 Family Instability	-0.048 (0.015)	-0.039 (0.025)	-0.108 (0.024)***	-0.036 (0.019)	-0.175 (0.019)***
Y1 Economic Hardship	-0.120 (0.025)***	-0.070 (0.025)**	-0.244 (0.022)***	-0.105 (0.019)***	-0.240 (0.018)***
Y3 Lack of Safety	-0.038 (0.027)	-0.063 (0.027)*	-0.161 (0.025)***	-0.045 (0.045)*	-0.174 (0.021)***
Y3 Family Instability	-0.030 (0.026)	-0.039 (0.026)	-0.057 (0.024)*	-0.029 (0.019)	-0.076 (0.019)***
Y3 Economic Hardship	-0.108 (0.027)***	-0.077 (0.027)**	-0.259 (0.024)***	-0.166 (0.021)***	-0.287 (0.019)***
Y5 Lack of Safety	-0.101 (0.025)***	-0.066 (0.026)*	-0.152 (0.023)***	-0.068 (0.019)***	-0.156 (0.021)***
Y5 Family Instability	-0.012 (0.026)	-0.034 (0.025)	-0.006 (0.024)	-0.029 (0.020)	-0.087 (0.020)***
Y5 Economic Hardship	-0.153 (0.031)***	-0.069 (0.025)**	-0.269 (0.022)***	-0.088 (0.019)***	-0.252 (0.018)***
Y9 Lack of Safety				-0.068 (0.019)***	-0.107 (0.019)***
Y9 Family Instability				-0.018 (0.019)	-0.067 (0.019)***
Y9 Economic Hardship				-0.103 (0.018)***	-0.220 (0.018)***

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Y=year.

^a Each outcome (Z-scores) examined in a separate model. Complete case analysis (FIML was not possible given no use of covariates).

APPENDIX, Table A4.4. Adjusted Relations Between Cumulative Adversities, Domains and Cognitive Outcomes ^a

	Y5 Sustained Attention Standardized β (SE)	Y5 Lack of Impulsivity Standardized β (SE)	Y5 PPVT-III Standardized β (SE)	Y9 Digit Span Standardized β (SE)	Y9 PPVT-III Standardized β (SE)
<i>Cumulative Adversities</i>					
Y1 Cumulative Index	-0.045 (0.026)	-0.021 (0.021)	-0.099 (0.023)***	-0.045 (0.021)*	-0.098 (0.018)***
Y3 Cumulative Index	-0.022 (0.026)	-0.012 (0.017)	-0.096 (0.025)***	-0.078 (0.024)***	-0.104 (0.021)***
Y5 Cumulative Index	-0.061 (0.027)*	-0.046 (0.028)	-0.118 (0.023)***	-0.031 (0.024)	-0.088 (0.022)***
Y9 Cumulative Index				-0.072 (0.020)***	-0.061 (0.019)***
<i>Domains ^b</i>					
Y1 Lack of Safety	-0.007 (0.025)	-0.012 (0.026)	-0.042 (0.022)	-0.012 (0.019)	-0.002 (0.017)
Y1 Family Instability	0.032 (0.026)	0.003 (0.027)	0.031 (0.023)	0.005 (0.020)	-0.035 (0.018)
Y1 Economic Hardship	-0.085 (0.026)***	-0.039 (0.027)	-0.125 (0.023)***	-0.055 (0.019)**	-0.097 (0.018)***
Y3 Lack of Safety	0.037 (0.028)	-0.019 (0.028)	-0.034 (0.024)	0.012 (0.023)	-0.035 (0.020)
Y3 Family Instability	-0.003 (0.026)	-0.020 (0.027)	-0.006 (0.023)	0.006 (0.020)	0.016 (0.018)
Y3 Economic Hardship	-0.061 (0.030)*	-0.040 (0.030)	-0.114 (0.025)***	-0.121 (0.023)***	-0.126 (0.021)***
Y5 Lack of Safety	-0.040 (0.026)	0.018 (0.026)	-0.019 (0.022)	0.000 (0.023)	-0.014 (0.021)
Y5 Family Instability	0.031 (0.026)	-0.041 (0.027)	-0.002 (0.022)	-0.008 (0.021)	-0.009 (0.018)
Y5 Economic Hardship	-0.074 (0.026)**	-0.057 (0.026)*	-0.154 (0.022)***	-0.042 (0.020)*	-0.115 (0.018)***
Y9 Lack of Safety				-0.038 (0.020)	0.001 (0.018)
Y9 Family Instability				0.017 (0.019)	0.004 (0.017)
Y9 Economic Hardship				-0.057 (0.020)**	-0.096 (0.018)***

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Y=year.

^a Each outcome (Z-scores) examined in a separate model. All models were a close fit according to fit statistics. Models adjusted for race, maternal education, gender and neonatal risk.

^b Domains of the same wave were examined in the same model.

APPENDIX, Table A4.5. Adjusted Longitudinal Relations Between Cumulative Indices and Cognitive Outcomes ^a

	Y5 Sustained Attention Standardized β (SE)	Y5 Lack of Impulsivity Standardized β (SE)	Y5 PPVT-III Standardized β (SE)	Y9 Digit Span Standardized β (SE)	Y9 PPVT-III Standardized β (SE)
<i>Model 1 – Covariates Only</i>					
Black (Ref = white)	-0.125 (0.031)***	-0.077 (0.033)*	-0.345 (0.026)***	-0.029 (0.025)	-0.191 (0.021)***
Hispanic	-0.005 (0.032)	-0.013 (0.034)	-0.366 (0.026)***	-0.103 (0.024)***	-0.086 (0.021)***
Other	-0.007 (0.026)	0.014 (0.027)	-0.065 (0.022)**	0.003 (0.019)	0.017 (0.016)
Female	0.175 (0.023)***	0.106 (0.024)	0.072 (0.020)***	-0.004 (0.019)	-0.069 (0.015)***
<High School Education	-0.110 (0.024)***	-0.077 (0.025)**	-0.254 (0.020)***	-0.119 (0.019)***	-0.161 (0.016)***
Neonatal Risk	-0.059 (0.023)*	-0.010 (0.024)	-0.035 (0.020)	-0.040 (0.018)*	-0.024 (0.015)
R2	0.065 (0.012)***	0.024 (0.007)***	0.201 (0.016)***	0.064 (0.010)***	0.463 (0.016)***
<i>Model 2 (covariates not shown)</i>					
Y1 Cumulative Index	-0.057 (0.026)*	-0.031 (0.027)	-0.114 (0.022)***	-0.036 (0.021)	-0.038 (0.017)*
R2	0.068 (0.012)***	0.025 (0.007)***	0.212 (0.016)***	0.065 (0.010)***	0.466 (0.016)***
<i>Model 3 (covariates not shown)</i>					
Y1 Cumulative Index	-0.056 (0.028)*	-0.029 (0.029)	-0.089 (0.023)***	-0.015 (0.012)	-0.024 (0.018)
Y3 Cumulative Index	-0.002 (0.030)	-0.007 (0.031)	-0.083 (0.025)***	-0.069 (0.025)**	-0.036 (0.020)
R2	0.068 (0.010)***	0.025 (0.007)***	0.218 (0.016)***	0.069 (0.010)***	0.467 (0.016)***
<i>Model 4 (covariates not shown)</i>					
Y1 Cumulative Index	-0.055 (0.028)*	-0.030 (0.029)	-0.090 (0.023)***	-0.020 (0.023)	-0.023 (0.019)
Y3 Cumulative Index	-0.001 (0.030)	-0.010 (0.031)	-0.077 (0.025)**	-0.069 (0.028)*	-0.035 (0.022)
Y5 Cumulative Index ^b	-0.055 (0.026)*	-0.033 (0.026)	-0.078 (0.025)**	0.016 (0.028)	-0.004 (0.022)
R2	0.067 (0.012)***	0.025 (0.007)***	0.216 (0.016)***	0.069 (0.010)***	0.467 (0.016)***
<i>Model 5 (covariates not shown)</i>					
Y1 Cumulative Index	-0.055 (0.028)*	-0.031 (0.029)	-0.090 (0.023)***	-0.020 (0.023)	-0.023 (0.019)
Y3 Cumulative Index	0.001 (0.030)	-0.007 (0.031)	-0.074 (0.025)**	-0.068 (0.027)*	-0.031 (0.022)
Y5 Cumulative Index ^b	-0.054 (0.026)*	-0.032 (0.026)	-0.074 (0.026)**	-0.014 (0.028)	-0.006 (0.022)
Y9 Cumulative Index ^c				-0.042 (0.021)*	-0.035 (0.022)
R2	0.067 (0.012)***	0.025 (0.007)***	0.216 (0.016)***	0.069 (0.010)***	0.468 (0.016)***

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$; Y=year. ^a All outcomes (Z-scores) examined in a single model. All models presented here are a good fit (RMSEA < 0.08; CFI > 0.95). ^b Estimates on Y5 outcomes are standardized correlations; ^c Estimates on Y9 cognitive outcomes are standardized correlations. Models 2-5 adjusted for race, maternal education, gender and neonatal risk.

APPENDIX, Table A4.6. Adjusted Longitudinal Relations Between Adversity Domains and Cognitive Outcomes ^a

	Y5 Sustained Attention Standardized β (SE)	Y5 Lack of Impulsivity Standardized β (SE)	Y5 PPVT-III Standardized β (SE)	Y9 Digit Span Standardized β (SE)	Y9 PPVT-III Standardized β (SE)
<i>Model 1</i>					
Y1 Lack of Safety	-0.017 (0.025)	-0.016 (0.026)	-0.053 (0.020)**	-0.009 (0.019)	0.027 (0.016)
Y1 Family Instability	0.028 (0.026)	0.001 (0.027)	0.013 (0.022)	0.000 (0.020)	-0.043 (0.017)*
Y1 Economic Hardship	-0.087 (0.026)***	-0.041 (0.027)	-0.117 (0.022)***	-0.039 (0.020)	-0.034 (0.017)*
R2	0.073 (0.012)***	0.027 (0.008)***	0.219 (0.016)***	0.066 (0.010)***	0.467 (0.016)***
<i>Model 2</i>					
Y1 Lack of Safety	-0.021 (0.025)	-0.009 (0.026)	-0.048 (0.021)*	-0.005 (0.019)	0.032 (0.016)*
Y1 Family Instability	0.029 (0.026)	0.008 (0.027)	0.025 (0.022)	0.010 (0.020)	-0.036 (0.017)*
Y1 Economic Hardship	-0.078 (0.028)**	-0.029 (0.029)	-0.080 (0.023)***	-0.006 (0.022)	-0.019 (0.018)
Y3 Lack of Safety	0.046 (0.028)	-0.016 (0.028)	-0.021 (0.023)	0.005 (0.023)	-0.016 (0.018)
Y3 Family Instability	0.007 (0.026)	-0.018 (0.027)	0.021 (0.021)	0.007 (0.020)	0.007 (0.016)
Y3 Economic Hardship	-0.040 (0.031)	-0.027 (0.032)	-0.115 (0.025)***	-0.109 (0.025)***	-0.041 (0.020)*
R2	0.075 (0.012)***	0.028 (0.008)***	0.229 (0.016)***	0.073 (0.011)***	0.470 (0.016)***
<i>Model 3</i>					
Y1 Lack of Safety	-0.023 (0.025)	-0.013 (0.026)	-0.051 (0.021)*	-0.007 (0.020)	0.032 (0.016)*
Y1 Family Instability	0.028 (0.026)	0.006 (0.027)	0.025 (0.022)	0.008 (0.021)	-0.036 (0.017)*
Y1 Economic Hardship	-0.078 (0.028)**	-0.030 (0.029)	-0.079 (0.023)***	-0.011 (0.022)	-0.019 (0.018)
Y3 Lack of Safety	0.045 (0.027)	-0.015 (0.028)	-0.015 (0.023)	0.002 (0.025)	-0.019 (0.020)
Y3 Family Instability	0.009 (0.026)	-0.017 (0.027)	0.022 (0.021)	0.010 (0.020)	0.009 (0.017)
Y3 Economic Hardship	-0.037 (0.031)	-0.020 (0.032)	-0.155 (0.025)***	-0.112 (0.026)***	-0.040 (0.021)
Y5 Lack of Safety ^b	-0.059 (0.025)*	0.021 (0.025)	-0.016 (0.023)	0.013 (0.025)	0.004 (0.020)
Y5 Family Instability ^b	0.016 (0.026)	-0.039 (0.026)	-0.026 (0.023)	-0.013 (0.021)	-0.002 (0.017)
Y5 Economic Hardship ^b	-0.058 (0.025)*	-0.045 (0.025)	-0.119 (0.022)***	0.019 (0.023)	-0.004 (0.018)
R2	0.074 (0.012)***	0.028 (0.008)***	0.229 (0.016)***	0.074 (0.011)***	0.471 (0.016)***

APPENDIX, Table A4.6. Adjusted Longitudinal Relations Between Adversity Domains and Cognitive Outcomes (continued) ^a

	Y5 Sustained Attention Standardized β (SE)	Y5 Lack of Impulsivity Standardized β (SE)	Y5 PPVT-III Standardized β (SE)	Y9 Digit Span Standardized β (SE)	Y9 PPVT-III Standardized β (SE)
<i>Model 4</i>					
Y1 Lack of Safety	-0.025 (0.025)	-0.013 (0.026)	-0.052 (0.021)*	-0.007 (0.019)	0.033 (0.016)*
Y1 Family Instability	0.028 (0.026)	0.006 (0.027)	0.027 (0.022)	0.009 (0.021)	-0.037 (0.017)*
Y1 Economic Hardship	-0.079 (0.028)**	-0.031 (0.029)	-0.080 (0.023)***	-0.012 (0.021)	-0.019 (0.018)
Y3 Lack of Safety	0.046 (0.027)	-0.014 (0.028)	-0.013 (0.023)	0.002 (0.025)	-0.015 (0.020)
Y3 Family Instability	0.009 (0.026)	-0.016 (0.027)	0.023 (0.021)	0.010 (0.020)	0.009 (0.017)
Y3 Economic Hardship	-0.034 (0.031)	-0.020 (0.032)	-0.115 (0.025)***	-0.114 (0.026)***	-0.041 (0.021)*
Y5 Lack of Safety ^b	-0.061 (0.025)*	0.021 (0.025)	-0.015 (0.023)	0.011 (0.025)	0.001 (0.019)
Y5 Family Instability ^b	0.016 (0.026)	-0.039 (0.026)	-0.025 (0.023)	-0.012 (0.021)	-0.002 (0.017)
Y5 Economic Hardship ^b	-0.059 (0.025)*	-0.045 (0.025)	-0.045 (0.025)	0.022 (0.023)	0.001 (0.018)
Y9 Lack of Safety ^c				-0.038 (0.020)	0.007 (0.021)
Y9 Family Instability ^c				0.010 (0.019)	-0.020 (0.020)
Y9 Economic Hardship ^c				-0.028 (0.019)	-0.028 (0.019)
R2	0.073 (0.012)***	0.028 (0.008)***	0.229 (0.016)***	0.075 (0.011)***	0.472 (0.016)***

*p<0.05; **p<0.01; ***p<0.001; Y=year.

^a All outcomes (Z-scores) examined in a single model. All models presented here are a good fit (RMSEA<0.08; CFI>0.95). Model adjusted for race, sex, maternal education and neonatal risk.

^b Estimates on Y5 cognitive outcomes are standardized correlations.

^c Estimates on Y9 cognitive outcomes are standardized correlations.

REFERENCES

1. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci* 2009;10:434–45.
2. Pechtel P, Pizzagalli DA. Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology* 2010;214:55–70.
3. Nelson CA, de Hann M, Thomas KM. *Neuroscience of Cognitive Development: The Role of Experience and the Developing Brain*. Hoboken, New Jersey: John Wiley & Sons Inc; 2006.
4. Nisbett RE, Aronson J, Blair C, et al. Intelligence: New findings and theoretical developments. *American Psychologist* 2012;67:130–59.
5. Brooks-Gunn J, Duncan GJ. The effects of poverty on children. *The Future of Children* 1997;7:55–71.
6. Centers for Disease Control and Prevention. Essentials for Childhood: Steps to create safe, stable, nurturing relationships and environments 2014. Retrieved 2015 Sept 8. Available from: http://www.cdc.gov/violenceprevention/pdf/essentials_for_childhood_framework.pdf.
7. Evans GW, Li D, Whipple SS. Cumulative risk and child development. *Psychological Bulletin* 2013;139:1342.
8. Sameroff AJ, Seifer R, Baldwin A, Baldwin C. Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development* 1993;64:80–97.
9. Anda RF, Felitti VJ, Bremner JD, et al. The enduring effects of abuse and related adverse experiences in childhood. *European archives of psychiatry and clinical neuroscience* 2006;256:174–86.
10. Bethell CD, Newacheck P, Hawes E, Halfon N. Adverse Childhood Experiences: Assessing The Impact On Health And School Engagement And The Mitigating Role Of Resilience. *Health Affairs* 2014;33:2106–15.
11. Noble KG, Tottenham N, Casey BJ. Neuroscience perspectives on disparities in school readiness and cognitive achievement. *The Future of Children* 2005;15:71–89.
12. Rutter M. Protective factors in children's responses to stress and disadvantage. *Annals of the Academy of Medicine, Singapore* 1979;8:324.
13. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and

household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine* 1998;14:245–58.

14. Alaimo K, Olson CM, Frongillo EA. Food insufficiency and American school-aged children's cognitive, academic, and psychosocial development. *Pediatrics* 2001;108:44–53.
15. Ayoub C, O'Connor E, Rappolt-Schlichtmann G, Vallotton C, Raikes H, Chazan-Cohen R. Cognitive skill performance among young children living in poverty: Risk, change, and the promotive effects of Early Head Start. *Early Childhood Research Quarterly* 2009;24:289–305.
16. Aro T, Poikkeus A-M, Eklund K, et al. Effects of Multidomain Risk Accumulation on Cognitive, Academic, and Behavioural Outcomes. *Journal of Clinical Child & Adolescent Psychology* 2009;38:883–98.
17. Burchinal MR, Roberts JE, Hooper S, Zeisel SA. Cumulative risk and early cognitive development: A comparison of statistical risk models. *Developmental Psychology* 2000;36:793–807.
18. Hall JE, Sammons P, Sylva K, et al. Measuring the combined risk to young children's cognitive development: An alternative to cumulative indices. *British Journal of Developmental Psychology* 2010;28:219–38.
19. Klebanov PK, Brooks-Gunn J. Cumulative, Human Capital, and Psychological Risk in the Context of Early Intervention: Links with IQ at Ages 3, 5, and 8. *Annals of the New York Academy of Sciences* 2006;1094:63–82.
20. Krishnakumar A, Black MM. Longitudinal predictors of competence among African American children: The role of distal and proximal risk factors. *Journal of Applied Developmental Psychology* 2002;23:237–66.
21. Liaw F-R, Brooks-Gunn J. Cumulative familial risks and low-birthweight children's cognitive and behavioral development. *Journal of Clinical Child Psychology* 1994;23:360–272.
22. Poehlmann J. Children's family environments and intellectual outcomes during maternal incarceration. *Journal of Marriage and Family* 2005;67:1275–85.
23. Biederman J, Milberger S, Faraone SV, et al. Family-environment risk factors for attention-deficit hyperactivity disorder: a test of Rutter's indicators of adversity. *Archives of General Psychiatry* 1995;52:464.
24. Crozier JC, Barth RP. Cognitive and academic functioning in maltreated children. *Children & Schools* 2005;27:197–206.
25. Mistry RS, Benner AD, Biesanz JC, Clark SL, Howes C. Family and social risk, and parental investments during the early childhood years as predictors of low-

- income children's school readiness outcomes. *Early Childhood Research Quarterly* 2010;25:432–49.
26. Pasco Fearon RM, Belsky J. Attachment and Attention: Protection in Relation to Gender and Cumulative Social -Contextual Adversity. *Child Development* 2004;75:1677–93.
 27. Lengua LJ, Honorado E, Bush NR. Contextual risk and parenting as predictors of effortful control and social competence in preschool children. *Journal of Applied Developmental Psychology* 2007;28:40–55.
 28. Evans GW, English K. The environment of poverty: Multiple stressor exposure, psychophysiological stress, and socioemotional adjustment. *Child Development* 2002;73:1238–48.
 29. Ackerman BP, Kogos J, Youngstrom E. Family instability and the problem behaviors of children from economically disadvantaged families. *Developmental Psychology* 1999;35:258-268.
 30. Guo G, Harris KM. The mechanisms mediating the effects of poverty on children's intellectual development. *Demography* 2000;37:431–47.
 31. Gershoff ET, Aber JL, Raver CC, Lennon MC. Income Is Not Enough: Incorporating Material Hardship Into Models of Income Associations With Parenting and Child Development. *Child Development* 2007;78:70–95.
 32. Schoon I, Jones E, Cheng H, Maughan B. Family hardship, family instability, and cognitive development. *Journal of Epidemiology and Community Health* 2012;66:716–22.
 33. De Bellis MD. Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy. *Development and Psychopathology* 2001;13:539–64.
 34. Watts English T, Fortson BL, Gibler N, Hooper SR, De Bellis MD. The psychobiology of maltreatment in childhood. *Journal of Social Issues* 2006;62:717–36.
 35. McEwen BS. Early life influences on life-long patterns of behavior and health. *Ment Retard Dev Disabil Res Rev* 2003;9:149–54.
 36. Danese A, McEwen BS. Adverse childhood experiences, allostasis, allostatic load, and age-related disease. *Physiology & Behavior* 2012;106:29–39.
 37. Gunnar M, Quevedo K. The Neurobiology of Stress and Development. *Annu Rev Psychol* 2007;58:145–73.
 38. Gunnar MR, Barr RG. Stress, early brain development, and behavior. *Infants &*

Young Children 1998;11:1–14.

39. McEwen BS. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology* 2008;583:174–85.
40. Hackman DA, Farah MJ. Socioeconomic status and the developing brain. *Trends in Cognitive Sciences* 2009;13:65–73.
41. Brown ED, Ackerman BP, Moore CA. Family adversity and inhibitory control for economically disadvantaged children: Preschool relations and associations with school readiness. *Journal of Family Psychology* 2013;27:443–52.
42. Johnson SB, Riley AW, Granger DA, Riis J. The Science of Early Life Toxic Stress for Pediatric Practice and Advocacy. *Pediatrics* 2013;131:319–27.
43. McEwen BS, Gianaros PJ. Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences* 2010;1186:190–222.
44. Shonkoff JP, Garner AS, The Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood, Adoption, and Dependent Care, and Section on Developmental and Behavioral Pediatrics, et al. The Lifelong Effects of Early Childhood Adversity and Toxic Stress. *Pediatrics* 2011;129:e232–46.
45. De Luca CR, Wood SJ, Anderson V, et al. Normative Data From the Cantab. I: Development of Executive Function Over the Lifespan. *Journal of Clinical and Experimental Neuropsychology (Neuropsychology, Development and Cognition: Section A)* 2003;25:242–54.
46. Cybele Raver C, McCoy DC, Lowenstein AE, Pess R. Predicting individual differences in low-income children's executive control from early to middle childhood. *Developmental Science* 2013;16:394–408.
47. Hackman DA, Gallop R, Evans GW, Farah MJ. Socioeconomic status and executive function: developmental trajectories and mediation. *Developmental Science* 2015;1-17.
48. Reichman NE, Teitler JO, Garfinkel I, McLanahan SS. Fragile families: Sample and design. *Children and Youth Services Review* 2001;23:303–26.
49. Campbell JM, Brown RT, Cavanagh SE, Vess SF, Segall MJ. Evidence-based Assessment of Cognitive Functioning in Pediatric Psychology. *Journal of Pediatric Psychology* 2008;33:999–1014.
50. Wechsler D. Wechsler Intelligence Scale for Children: WISC-IV ®. 4 ed. San Antonio, TX: Harcourt Assessment; 2003.

51. Dunn LM. Peabody Picture Vocabulary Test. 3rd ed. Circle Pines, MN: American Guidance Service; 1997.
52. Roid GH, Miller LJ. Leiter International Performance Scale-Revised. Wood Dale, IL: Stoelting Co; 1997.
53. Straus MA, Hamby SL, Finkelhor D, Moore DW, Runyan D. Identification of child maltreatment with the Parent-Child Conflict Tactics Scales: Development and psychometric data for a national sample of American parents. *Child Abuse & Neglect* 1998;22:249–70.
54. Straus MA, Field CJ. Psychological aggression by American parents: National data on prevalence, chronicity, and severity. *Journal of Marriage and Family* 2003;65:795–808.
55. Straus MA, Stewart JH. Corporal punishment by American parents: National data on prevalence, chronicity, severity, and duration, in relation to child and family characteristics. *Clin Child Fam Psychol Rev* 1999;2:55–70.
56. Sweet J, Bumpass L, Call V. The Design and Content of the National Survey of Families and Households. University of Wisconsin--Madison. Center for Demography and Ecology; 1988. Retrieved 2015 Sept 2. Available from: <http://www.ssc.wisc.edu/cde/nsfhwf/nsfh1.pdf>.
57. Lloyd S. The Effects of Violence on Women's Employment. *Law and Policy* 1997;19:139–67.
58. Waldfogel J, Craigie TA, Brooks-Gunn J. Fragile families and child wellbeing. *The Future of Children* 2010;20:87.
59. Cavanagh SE, Huston AC. Family instability and children's early problem behavior. *Social Forces* 2006;85:551–81.
60. Craigie T-AL, Brooks-Gunn J, Waldfogel J. Family structure, family stability and outcomes of five-year-old children. *Families, Relationships and Societies* 2012;1:43–61.
61. Kessler RC, Andrews G, Mroczek D, Ustun B, Wittchen HU. The World Health Organization Composite International Diagnostic Interview Short -Form (CIDI -SF). *International journal of methods in psychiatric research* 2005;7:171–85.
62. Bureau UC. Survey on Income and Program Participation. Washington, DC: 1996.
63. Wechsler D. Wechsler Adult Intelligence Scale - revised (WAIS-R Manual). Harcourt Brace Jovanovich; 1981.
64. Schlomer GL, Bauman S, Card NA. Best practices for missing data management

- in counseling psychology. *Journal of Counseling Psychology* 2010;57:1–10.
65. Little TD, Jorgensen TD, Lang KM, Moore EWG. On the Joys of Missing Data. *Journal of Pediatric Psychology* 2014;39:151–62.
 66. Enders CK. *Applied Missing Data Analysis*. New York: Guilford Publications; 2010.
 67. Muthén BO, Muthén LK. *Mplus User's Guide*. Seventh Edition. Los Angeles: 2012.
 68. Bollen KA. *Structural Equations with Latent Variables*. Canada: John Wiley and Sons, Inc; 1989.
 69. Little TD. *Longitudinal Structural Equation Modeling*. New York: The Guilford Press; 2013.
 70. De Bellis MD. The Psychobiology of Neglect. *Child Maltreatment* 2005;10:150–72.
 71. Evans GW. A multimethodological analysis of cumulative risk and allostatic load among rural children. *Developmental Psychology* 2003;39:924–33.
 72. Evans GW, Kim P. Multiple risk exposure as a potential explanatory mechanism for the socioeconomic status-health gradient. *Annals of the New York Academy of Sciences* 2010;1186:174–89.
 73. Fomby P, Cherlin AJ. Family instability and child well-being. *American Sociological Review* 2007;27:181-204.
 74. Blair C, Granger DA, Willoughby M, et al. Salivary Cortisol Mediates Effects of Poverty and Parenting on Executive Functions in Early Childhood. *Child Development* 2011;82:1970–84.
 75. Harding JF. Increases in maternal education and low-income children's cognitive and behavioral outcomes. *Developmental Psychology* 2015;51:583–99.
 76. Carneiro P, Meghir C, Parey M. Maternal Education, Home Environments, and the Development of Children and Adolescents. *Journal of the European Economic Association* 2012;11:123–60.
 77. Gunn JB, Klebanov PK, Duncan GJ. Ethnic differences in children's intelligence test scores: Role of economic deprivation, home environment, and maternal characteristics. *Child Development* 1996;67:396-408.
 78. Doyle O, Harmon CP, Heckman JJ, Tremblay RE. Investing in early human development: Timing and economic efficiency. *Economics & Human Biology* 2009;7:1–6.

79. Martin A, Razza RA, Brooks-Gunn J. Sustained attention at age 5 predicts attention-related problems at age 9. *International Journal of Behavioral Development* 2012;36:413–9.
80. Petersen SE, Posner MI. The Attention System of the Human Brain: 20 Years After. *Annu Rev Neurosci* 2012;35:73–89.
81. Rueda MR, Fan J, McCandliss BD, et al. Development of attentional networks in childhood. *Neuropsychologia* 2004;42:1029–40.
82. Mezzacappa E. Alerting, orienting, and executive attention: Developmental properties and sociodemographic correlates in an epidemiological sample of young, urban children. *Child Development* 2004;75:1373–86.
83. Dahl GB, Lochner L. The Impact of Family Income on Child Achievement: Evidence from the Earned Income Tax Credit. *American Economic Review* 2012;102:1927–56.
84. Centers for Disease Control and Prevention. Adverse childhood experiences reported by adults---five states, 2009. *MMWR Morbidity and mortality weekly report* 2010;59:1609.
85. Anda RF, Butchart A, Felitti VJ, Brown DW. Building a Framework for Global Surveillance of the Public Health Implications of Adverse Childhood Experiences. *American Journal of Preventive Medicine* 2010;39:93–8.
86. Paulsell D, Avellar S, Martin ES, Del Grosso P. Home visiting evidence of effectiveness review: Executive summary 2010; Retrieved 2015 Sept 8; Available from: http://homvee.acf.hhs.gov/HomVEE_Executive_Summary.pdf.
87. Camilli G, Vargas S, Ryan S, Barnett WS. Meta-analysis of the effects of early education interventions on cognitive and social development. *Teach Coll Rec* 2010; 112:579-620.

CHAPTER 5

MEDIATORS OF EARLY ADVERSITY DOMAINS ON CHILD COGNITIVE DEVELOPMENT

INTRODUCTION

Adverse experiences refer to events or exposures outside of a child's control that typically create excessive demands or threats to the child but are preventable or amenable to change. These include experiences of abuse, neglect, family instability, parental mental illness, parental substance abuse, parental incarceration, domestic violence, low socioeconomic status, and exposure to neighborhood violence. Adverse experiences often co-occur (1,2), and there is good evidence that as the number of adverse experiences in a child's life increases, there are declines in cognitive outcomes, academic achievement, social competencies and self-regulatory behavior (3).

The underlying pathways explaining the relationship between multiple adverse experiences and developmental outcomes, however, are less clear. In order to better understand the mechanisms through which multiple adverse experiences influence development and to design more efficient, effective interventions, some have recommended evaluating groups of adversities, or adversity domains, in relation to developmental outcomes and other mediating factors (3,4). The current study builds upon this recommendation, evaluating whether characteristics of the home environment – maternal warmth and the availability of reading materials – mediate the relation between domains of adverse experiences and child cognitive development.

Multiple Adverse Experiences and Child Cognitive Development

General cognitive ability and executive function are important early foundations of school readiness and academic achievement (5,6). General cognitive ability (i.e., intellectual capacity, IQ) refers to general reasoning and thinking ability and is associated

with one's ability to plan and solve problems (6). Executive functions are distinct neurocognitive processes including sustained attention, working memory, and impulse control (7,8). These processes are critical for learning because they allow children to hold information in memory, focus attention and avoid distraction (9).

General cognitive ability and executive function are shaped by social context (6,10). A prior review of the literature showed that children exposed to multiple adverse experiences had impaired cognitive functioning (see Chapter 3). Specifically, children exposed to multiple adverse experiences demonstrated lower scores on assessments of general cognitive ability, and this effect has been observed from infancy throughout adolescence. Aspects of executive function, which are known to improve throughout childhood and adolescence, were also negatively impacted by multiple adverse experiences. Children who experienced multiple adversities showed less improvement in executive function over time relative to children with fewer exposures.

Adversity Domains

The majority of studies that have evaluated the relations between multiple adverse experiences and child cognitive outcomes have done so using a cumulative index, which is created by dichotomizing each adversity (1= exposed and 0 = unexposed) and then summing the total number of exposures into a single measure. There are several strengths to this approach. This method preserves statistical power in small sample sizes and avoids issues of collinearity by combining multiple, highly correlated measures into a single metric (3). However, the cumulative index also weights each adversity equally, and this limits the ability to make inferences about the salience of specific adverse

exposures or the relations between exposures. These insights are important for informing intervention targets (3).

Evans, Whipple and Li (2013) recommend the use of adversity domains as an alternative to the cumulative index approach described above. Adversity domains are created by aggregating adversities of a similar type or context into groups. This approach leverages the advantages of a cumulative measure while also providing additional insight into the salience of particular domains or relations between domains and other mediating factors (3).

Few studies have used this domain-based approach to evaluate the relation between multiple adverse experiences and child cognitive development.

Among those that have, one study showed significant effects on children's cognitive ability at ages three, five and eight for only a human capital domain measured in infancy, comprising maternal education, employment and welfare status, but not a psychological domain (also measured in infancy; made up of low social support, maternal depression, and stressful life events) (4). Another study showed that economic hardship, but not family instability (defined by the number of changes in parental relationship status, was significantly associated with cognitive ability in five year olds (11). However, a study evaluating the influence of poverty and family chaos and instability among low-income children's inhibitory control, showed that family chaos and instability explained more of the variance in inhibitory control than did poverty (12).

In our prior work, we examined the relations between three different adversity domains and child cognitive outcomes. These domains included economic hardship (living at or below the federal poverty level, food insecurity and housing insecurity),

family instability (parental relationship instability, father incarceration, and maternal depression), and lack of safety (severe corporal punishment, severe psychological aggression, neglect, exposure to domestic violence, and community violence). Each domain consisted of an aggregate score of the adverse experiences within that domain. We evaluated the relations between these three domains measured at four ages across early and middle childhood and measures of child attention, working memory, and verbal ability (a proxy measure for general cognitive ability) assessed at ages five and nine years. We found the most salient effects for the economic hardship domain. Economic hardship during infancy negatively predicted sustained attention and verbal ability at age five, and economic hardship at age three negatively predicted working memory/attention and verbal ability at age nine. Although the effects of economic hardship were most striking, higher scores in the lack of safety and family instability domains in infancy also negatively impacted verbal ability (see Chapter 4 for study details).

Although studies using adversity domains are limited, as seen above, they do demonstrate that different types of adversities may differentially impact cognitive development. The current study builds upon our prior work, examining the three adversity domains described above – economic hardship, lack of safety and family instability – and the potential mediating roles of home environment and maternal warmth. Moreover, we examine these relationships separately for boys and girls, given the known differences in their patterns and timing of cognitive development.

Mediating Role of the Home Environment

Few domain-based studies have examined factors that mediate the relationship between adverse experiences and cognitive outcomes. Tests of mediating effects with different constructs are necessary to discern if there are one or more shared underlying mechanisms explaining the relations between adversity domains and cognitive outcomes (3).

Several theories provide insight into possible mediating pathways between adversity domains and child cognitive development. Bronfenbrenner's bioecological theory of human development provides an overarching perspective and posits that development is shaped by the interactions, *or proximal processes*, between an individual and the most immediate, frequently experienced aspects of their environment over time (13). The confluence of multiple adversities may be more likely to disrupt such processes, especially those necessary for healthy child development (i.e., parent/child interactions), whereas exposure to any single adversity may be more easily overcome through alternative processes (3,14).

Research to identify factors that may explain the causal mechanisms by which adversities exert their effects on cognitive development have focused on the known supports for healthy child development that may be disrupted by adversity.

Lack of stimulating home environments (including learning and literacy activities and use of diverse vocabulary) is one of the more consistent mediators observed between adversities related to economic hardship and cognitive outcomes, particularly for outcomes of general cognitive ability (15-19). Specifically, the lack of stimulating home environments explains, in part, the relation between poor children and lower cognitive

scores. This mediating pathway is supported by substantial research showing that poverty may limit the capacity of families to invest in stimulating home environments (i.e., books, activities) as well as other resources and services that benefit child health, including nutrition, housing, health care, and child care (20-22).

In addition to stimulating home environments, research has also identified parenting behaviors and parent/child interactions as mediators of the relations between multiple adversities and cognitive outcomes. Research focusing specifically on the effect of income and economic hardship on cognitive development has shown that stress associated with financial need may interfere with parents' abilities to allocate time and energy for interacting positively with their children (22,23). A handful of other studies have examined the mediating role of parenting characteristics on outcomes of executive function. For example, the negative relationship between a cumulative index of adversities (including single mother, unemployed mother, living in poverty, receipt of public assistance, maternal depression, and inability to meet basic needs) in the first year of life and a latent construct of attention and behavioral regulation at age three was mediated by lack of maternal warmth (24). Another, person-centered approach showed that children of low-income families with unmarried parents or a single mother had poorer executive function at 36 months, and lack of maternal positive engagement mediated this relationship (25). Among preschoolers, family income and a separate cumulative index of adversities (including low maternal education, single parent, adolescent parent, residential instability, divorce, household density, negative life events and maternal depression) was associated with poorer executive control, and effects were

mediated by less parental limit setting and scaffolding; there was no significant mediation of warmth, negativity, or responsiveness (26).

Of interest in the current study are the mediating roles of cognitive stimulation (measured by the availability of reading materials) and maternal warmth in the relations between our proposed adversity domains and child cognitive outcomes. Although there is substantial evidence that cognitive stimulation mediates the relation between economic hardship and cognitive development through family investment strategies (20-22), less is known about whether cognitive stimulation also mediates the relation between the lack of safety and family instability domains. We hypothesize that cognitive stimulation will mediate the relation between the economic hardship domain and child cognitive outcomes, and we explore whether there is any relation between the lack of safety and family instability domains on cognitive stimulation.

We hypothesize that exposure to multiple adverse experiences will negatively affect maternal warmth, thus negatively affecting cognitive outcomes in children. This hypothesis is supported by research on the neurobiology of stress and attachment. Adversity not only interferes with parenting behaviors, but it is also stressful to children, evidenced by heightened stress reactivity among children exposed to multiple adversities (27,28), unsafe environments caused by violence and maltreatment (29-31), and poverty (32,33). Research from animal and human models suggest that overproduction of cortisol in response to chronic stress and the underproduction of cortisol that may arise from severe deprivation can inhibit neurogenesis in the hippocampus and the prefrontal cortex, which are important brain regions for learning, memory, and executive function (31,34-36). More responsive parenting has been shown to yield more securely attached children,

and more securely attached children are less reactive to acute stressors (35,37,38). However, multiple adversities in the home environment can interfere with a parent's capacity to demonstrate warmth. A study of two year olds found that greater household risk predicted less positive parenting, which in turn predicted higher cortisol levels in children and worse executive function (33). All three domains of adversity in this study can influence both parenting behaviors and children's stress level, and therefore, we hypothesized that maternal warmth will mediate the relation between all three domains and all cognitive outcomes.

Gender Differences

Despite developmental differences in early cognitive development between boys and girls, little is known about whether gender moderates the effects of early adversity or parent/child interactions on cognitive outcomes. In early childhood, girls consistently outperform boys on measures of attention and verbal ability, though boys eventually catch up to girls (39,40). These findings were supported by our previous analysis of the same sample as the current study; girls scored significantly higher than boys on measures of sustained attention, lack of impulsivity, and verbal ability at age five, but there were no significant differences in verbal ability or working memory and attention at age nine (Chapter 4).

Evidence for a moderating effect of gender on the relations between adversity, the home environment, and cognitive development comes from two studies that showed gender moderated the effect of an Early Head Start intervention on the relation between parental stress and children's vocabulary in early childhood (40). In the first study, the

intervention was protective for girls, but not for boys, and in the second study, the intervention boosted girls' vocabulary, regardless of the level of parenting stress and buffered the effect of parenting stress on boys. A third study found that boys exposed to high adversity performed worse than girls on measures of attention. This effect was moderated by attachment style; boys without secure attachment to their mothers performed significantly worse than girls without secure attachment (39). These studies support one hypothesis that boys may be more affected by disruptions in parent-child interactions that occur in the context of adversity whereas girls may be affected by adversity directly (40). Although, there is little research in this area, there is some reason to suspect that the cognitive stimulation and maternal warmth may be more significant mediators of the relation between adversity and cognitive outcomes for boys. The current study aims to add to this body of research by exploring whether gender moderates the mediating role of the home environment in the relation between adversity domains and cognitive development.

The Current Study

The current study utilizes publically available data from a longitudinal cohort of children from birth to age nine. We build upon our earlier work that examined the influence of three different adversity domains (economic hardship, lack of safety, and family instability) on child cognitive outcomes. This study evaluates whether two characteristics of the home environment – maternal warmth and availability of reading materials – mediate the relations between the adversity domains and cognitive outcomes. We focus specifically on outcomes of receptive vocabulary (a proxy measure for

cognitive ability), sustained attention, lack of impulsivity and working memory/attention. We hypothesized that availability of reading materials would mediate the relation between the economic hardship domain and all cognitive outcomes, and we explored the mediating role of availability of reading materials in the relation between the lack of safety and family instability domains on all cognitive outcomes. We also hypothesized that maternal warmth would mediate the relation between all three adversity domains and all three cognitive outcomes. Additionally, we evaluated whether gender moderated these effects. Given the paucity of research on gender differences, the current study aimed to add to the existing literature base in this area, but no specific hypotheses were made.

METHODS

Sample

The Fragile Families and Child Wellbeing (FFCW) Study follows a birth cohort of 4789 children born between 1998 and 2000 from 20 large U.S. cities (population >200,000) (41). The sampling of individuals occurred in three stages: first cities, then hospitals within cities, then births within hospitals. Children born to unmarried parents were oversampled ($n=3647$ vs. $n=1141$ children born to married parents) in order to be representative of non-marital births (see Reichman, Teitler, Garfinkel, & McLanahan for more details on study design).

As part of the core study, biological mothers were interviewed in the hospital within 48 hours of the focal child's birth, and biological fathers were interviewed by phone soon after. Both biological parents were interviewed again by phone when the child was one, three, five and nine years of age. The current paper also draws upon the

In-Home Longitudinal Study of Preschool-Aged Children, a sub-study in which biological mothers who participated in the core study at years three and five were invited to participate. For the sub-study, primary caregivers participated in an additional interview and a home visit when the child was three and five years old. During the home visit, an investigator observed the home environment and directly assessed the child. At age nine, the in-home sub-study was integrated with the core study such that all participants started with a home visit and then completed the primary caregiver and core surveys.

From the original 20-city sample (N=4789), 132 (3%) of families were ineligible for the current study because children had conditions likely to influence cognitive outcomes, including: total or partial blindness, total or partial deafness, Down's syndrome, cerebral palsy, mental retardation or other developmental delay, and autism. Additionally, 1391 (29%) families were excluded because the child did not have at least one cognitive outcome measurement at age nine. To minimize measurement bias, the sample was also limited to those in which the biological mother was the primary caregiver (as opposed to father or other guardian; 290 excluded (6%)). The final analytic sample (N=2976) was slightly more advantaged than those excluded from the final analysis (35% below federal poverty line vs. 39%; 37% of mothers with greater than high school education vs. 31%), more likely to be non-Hispanic black (50% vs. 43%) and less likely to be non-Hispanic white (20% vs. 23%), Hispanic (27% vs. 29%), or other race (3.4% vs. 5.3%). Forty percent of mothers were not married or cohabitating at the time of the child's birth, consistent with the oversampling of non-marital births planned for in the study design. However, given the differences between the original and analytical

samples, the final analytical sample can no longer be considered fully representative of non-marital births in large U.S. cities. Descriptive characteristics of the final sample are shown in Table 5.1.

Measures

Cognitive Outcomes. The FFCW study collected well-established measures of cognitive functioning in children (42). The following assessments were conducted with the focal child in the child's home by a field interviewer during the in-home assessments.

Wechsler Intelligence Scale for Children (WISC-IV), Digit Span subtest: The WISC-IV is an intelligence test for children ages 6-16 years designed to measure child cognitive function. The Digit Span subtest of the WISC-IV specifically measures the child's auditory short-term memory, sequencing skills, attention, and concentration. At age nine, children heard a sequence of numbers and were asked to repeat the numbers either forward or backwards. Scores were age-normed (standard score of $M=10$, $SD=3$). The subtest has high internal consistency ($\alpha = 0.92$) and high test-retest reliability ($r = 0.89$) (43).

Child Peabody Picture Vocabulary Test (PPVT)-III: The PPVT-III measures receptive vocabulary and screens for verbal ability. At ages five and nine, an interviewer read a word and asked the child to identify the corresponding picture (among a set of four pictures) on an easel. Scores were age-normed (standard score of $M = 100$, $SD = 15$). The PPVT-III has high internal reliability ($\alpha = 0.93$) and test-retest reliability ($r=0.95$) (44).

Sustained Attention and Lack of Impulsivity: The Leiter International

Performance Scale —Revised measures children’s ability to maintain attention to a specific stimulus over time. At age five, children were shown a picture booklet with a variety of objects placed throughout the page. There was a target object at the top of the page, and children were asked to put a line through as many of the matching target pictures as possible within the allotted time, without erroneously crossing out non-target objects. Average performance across four trials yielded two attention scores. The number of correct responses reflected the child’s *sustained attention* whereas the number of incorrect responses (reverse coded) reflected *lack of impulsivity*. Scores were age-normed (standard score of $M=10$, $SD = 3$). The task has high internal reliability ($\alpha = 0.83$) and test–retest reliability ($r = 0.85$) for children 4–5 years of age (45).

Adverse Experiences. Biological mothers reported on the following measures during the core and the primary caregiver interviews at baseline and ages one, three, five and nine. In the first year of life, some of the adversity measures were collected at either baseline or age one, and therefore, these waves were combined and collectively referred to as *infancy*. Where possible, the same adversity measures were used at each wave of data collection. However, as described below there were a few instances where measures differed across waves, or where adversities were not measured at all waves. All of the adversities were dichotomized such that 1 = exposed, and 0 = unexposed based on theoretical cut-points.¹⁶

¹⁶ Maternal drug and alcohol use were also explored as adverse exposures in this study. However, too few respondents in the sample (<1%) reported these exposures, and therefore, these measures were not included in the final analysis.

Severe Psychological Aggression: The Parent-Child Conflict Tactics Scales (PCCTS) measures child maltreatment and nonviolent modes of discipline by parents (46). The 5-item psychological aggression subscale of the PCCTS measures verbal and symbolic acts by the parent intended to cause a child psychological pain or fear. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often they had done the following to the child in the past year: shouted, yelled or screamed at; threatened to spank or hit but didn't actually do it; swore or cursed at; called him or her dumb, lazy or some other name like that; said they would send them away or kick them out of the house. Ordinal responses included "never," "once," "twice," three to five times," etc. Among national samples, approximately 90% of parents report one or more forms of psychological aggression (also reflected in the current study population) (47). However, more severe forms of aggression (swore or cursed at, called dumb or lazy, or threatened to kick out of the house) are less common. Children of mothers who reported that at least one of these *more severe acts* occurred at least once in the last year were categorized as exposed to psychological abuse (prevalence score cut-offs for all PCCTS measures described in Straus et. al., 1998).

Severe Corporal Punishment: Corporal punishment was assessed with a 5-item subscale of the PCCTS. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often in the past year they: spanked the child on the bottom with a bare hand; hit the child on the bottom with something like a belt, hairbrush, a stick or some other hard object; slapped the child on the hand, arm or leg; pinched the child; and shook the child. Ordinal responses included "never," "once," "twice," three to five times," etc. Spanking the child and slapping the child on the arm or leg are

considered to be more widely accepted forms of corporal punishment, whereas the other three acts are considered to carry higher risks and be less widely accepted, thus indicating more severe corporal punishment (48). Children of mothers who reported that at least one of these three *more severe acts* occurred at least once in the last year were categorized as exposed to severe corporal punishment.

Child Neglect: Child neglect was assessed with a 5-item subscale of the PCCTS. During the primary caregiver survey at ages three, five and nine, biological mothers were asked how often they: had to leave the child home alone, even when they thought an adult should be with the child; were so caught up with her own problems that they were not able to show or tell the child they loved him/her; were not able to make sure that the child got the food he/she needed; were not able to make sure the child got to a doctor or hospital when needed; had drinking or drugs interfere with taking care of the child. Ordinal responses included “never,” “once,” “twice,” three to five times,” etc. Children of mothers who reported that at least one of these acts occurred at least once in the last year were categorized as exposed to child neglect.

Intimate Partner Violence (IPV): During the core surveys when the child was one, three, five and nine, biological mothers were asked to think about their relationship with the child’s father, or their current partner. For each existing romantic relationship (either with the biological father or a current partner), they were asked previously validated questions (49,50): 1) How often does he slap or kick you?; 2) How often does he hit you with a fist or object that could hurt you?; and 3) How often does he try to make you have sex or do sexual things that you don’t want to? For any relationship with the father (romantic or not) as well as for existing relationships with another current partner,

mothers were also asked, “Have you and the father (or current partner) been in a physical fight in front of the child in the time since the last interview?” If mothers answered “sometimes” or “often” to any of the first three questions, or, “yes” to the last question, they were categorized as experiencing IPV for that time period.

Exposure to Community Violence: Different measures were used to assess exposure to community violence at infancy and the later waves. At infancy, biological mothers were asked during the baseline core survey how safe the streets around their house were at night (very safe, safe, unsafe or very unsafe). Responses of unsafe or very unsafe were categorized as exposure. During the primary caregiver surveys at ages three, five and nine, biological mothers were asked about their own exposure to violence in their neighborhood in the past year. Three questions assessed whether the primary caregivers saw someone get hit, punched, slapped or beaten up by someone else; if they saw someone get attacked with a weapon like a knife or a bat; and if they saw someone get shot. Ordinal responses ranged from never to more than ten times. Exposure to community violence at these waves was defined as at least one exposure to any of these three items.

Parental Relationship Instability: Relationship instability was defined as a change in parental relationship status since the child’s birth (51,52). Prior studies using data from the FFCW Study have shown that children with stable family structures (whether married, cohabitating, or single parents) had better outcomes than children with unstable family structures (characterized by a parent’s partial presence) (53). During the core surveys at baseline and when the child was three, five and nine, biological mothers were asked about their relationship with the biological father. Responses were

categorized into: married, cohabitating or single. During the infancy wave, adversity was simply classified as having a single parent family structure at the time of the child's birth, as opposed to a married or cohabitating family structure. For the remaining waves, stability was defined as having the same parent structure since the previous wave or moving from a cohabitating relationship to a married relationship since the previous wave. Moving from a married relationship to a cohabitating or single status, or moving from cohabitating relationship to single status was categorized as unstable.

Maternal Depression: The Composite International Diagnostic Interview (CIDI) is a standardized instrument for assessing mental disorders based on DSM-IV criteria. The short form (SF) of the CIDI interview takes a portion of the full set of CIDI questions and generates from the responses the probability that the respondent would be a case, if given the full interview (54). When the child was one, three, five and nine, biological mothers were asked all of the essential CIDI-SF questions necessary to classify a major depressive episode. Mothers who met established criteria were classified as probable cases for maternal depression.

Father Incarceration: Father incarceration was determined from both the mother report on the core surveys when the child was one, three, five and nine, and from information collected by interviewers in the field. Mothers were asked whether the father was currently in jail. Fathers were categorized as currently in jail if mothers or interviewers indicated this to be the case.

Living in Poverty: The income to needs ratio adjusts family income by the number of adults and children in the household, using the official poverty thresholds. Absolute poverty is measured by having a poverty ratio less than one. Family income and

family size were collected from the biological mother during the core survey at baseline and ages three, five and nine. Living in poverty was categorized as living below the federal poverty level.

Housing Insecurity: During the core surveys at ages one, three, five and nine, biological mothers were asked four questions derived from the Survey of Income and Program Participation and the New York City Social Indicators Survey (55), including whether they: had been evicted from their home in the past 12 months; stayed in a shelter/car or abandoned vehicle; did not pay full rent or mortgage; or if they had moved in with other people because of financial problems. Mothers responding “yes” to at least one of these questions were categorized as experiencing housing insecurity.

Food Insecurity: During the core survey at age one, the primary caregiver survey at age three, and the core surveys at ages five and nine, biological mothers were asked about whether, in the past 12 months, they were ever hungry but could not afford to buy more food. Mothers who responded “yes” to this question were characterized as experiencing food insecurity.

Adversity Domains: As discussed, grouping adverse exposures into domains allows analysis of potentially different effects of types of adversities. Three theoretically determined adversity domains, *lack of safety*, *instability* and *economic hardship*, were used to group exposures by context. The *lack of safety* domain included two adversities at infancy (exposure to community violence and maternal IPV), and five adversities at ages three, five and nine (severe psychological aggression, severe corporal punishment, neglect, community violence and maternal IPV). The *instability* domain included three adversities at all four waves: exposure to parental relationship instability (or single parent

status at infancy), maternal depression, and father incarceration. The *economic hardship* domain included the same three adversities at all four waves: living below the poverty level, food insecurity and housing instability. Each domain was created by summing the total number of adverse exposures within that domain.

Characteristics of Home Environment (Mediating Variables). Two characteristics of the home environment were assessed during the home visits at ages three and five years.

Maternal Warmth: Maternal warmth was determined from five yes/no items of the observational Home Observation for Measurement of the Environment (HOME) scales (56,57) and the Homelife Interview (58). Items included: parent talks with child twice during the visit, parent answers child's questions orally, parent praises child twice during the visit, parent voices positive feelings to child, and parent caresses, kisses, or hugs child. A maternal warmth score was created by summing the five items from each wave ($\alpha = 0.71$ for age three, $\alpha = 0.63$ for age five).

Availability of Reading Materials: At age three, mothers reported on the number of adult books in the house (none, 1-9, 10-20, or more than 20) and the number of books for the child in the house (none, 1-2, 3-4, or more than 4). At age five, mothers reported on the number of books in the house (none, 1-9, 10-20, or more than 20) and the number of books and games to help the child learn the alphabet (none, 1-2, 3-4, or more than 4). An availability of reading materials score was created for each year by summing the two items.

Control Variables. During the baseline core survey, biological mothers reported on the following control variables: maternal race/ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, and other), child sex (male/female), maternal education (high school education or less vs. greater than high school education), and neonatal risk. Neonatal risk was a constructed variable denoting whether the child was born with low birth weight (<2500g) *or* as a part of a multiple birth (twin). Publicly available data from the FFCW Study recorded all children born as a twin as missing in birth weight. Since twins are more likely to be born low birth weight, the two variables were combined into a single measure of neonatal risk. Even if a child was part of a multiple birth, only one focal child was included in the study.

In addition to these variables, prenatal substance use (drinking, smoking, drug use), maternal age, and maternal cognitive ability (similarities subtest from the Wechsler Adult Intelligence Scale – Revised (59)) were also included as controls in preliminary analyses. However, these variables were not used in the final analyses. The prenatal substance abuse variables and maternal age were dropped due to lack of significance in relation to all cognitive outcomes in both bivariate and multivariate analyses. Maternal cognitive ability was dropped because this variable was moderately correlated with maternal education ($r = 0.37$), and maternal education was used as a proxy for maternal cognitive ability. Sensitivity analysis demonstrated no substantive differences in the relationships between adversities and cognitive outcomes between the models that included both maternal education and cognitive ability and those that did not control for mother cognitive ability.

Missing Data Analysis

As shown in Table 5.1, data were missing for less than 1% of cases for each of the control variables, 38% - 44% for the age five cognitive outcomes, less than 1% for the age nine cognitive outcomes, 2% - 28% for the adversity domains, and 24-48% for the mediating variables. Data were missing mostly due to attrition rather than item non-response. Missingness due to attrition was less than 7% for each wave of the core study, 24% for the age three primary caregiver interview, 26% for the age five primary caregiver interview, and 38% for the in-home assessments. In order to determine systematic reasons for missing data, the relationships between observed variables and missing values were examined empirically (56). In general, those who participated in the core and primary caregiver interviews were more advantaged and less likely to be Hispanic compared to those who did not participate, consistent with earlier trends describing the analytic sample. However, those who participated in the in-home assessments were *less* advantaged than those who did not participate.

Missing data were classified as missing at random (MAR) because factors associated with missingness were observed in the data set (such as race, education, poverty level and marital status). While the MAR mechanism introduces bias, this bias is recoverable with modern missing data methods (60). Full-information maximum likelihood (FIML) was used to handle missing data in these analyses. FIML is a model-based approach that uses all of the available data to estimate the parameters of the statistical model in the presence of missing data and produces unbiased estimates of model parameters and standard errors (60,61). In this sample, nearly all variables missing more than 5% of data were correlated with at least one other variable used in the

model or one of nine auxiliary variables (variables that were peripheral to the substantive analysis but provided information about missingness) at a correlation greater than 0.30.

Analytic Approach

We first examined the means and standard deviations for continuous variables, frequencies for categorical or dichotomous variables, correlations between study variables, and univariate path models regressing each cognitive outcome on the key study variables using in Mplus 7.3 (62). Linear assumptions between the adversity domains, mediating variables and cognitive outcomes were examined graphically in Stata 13.

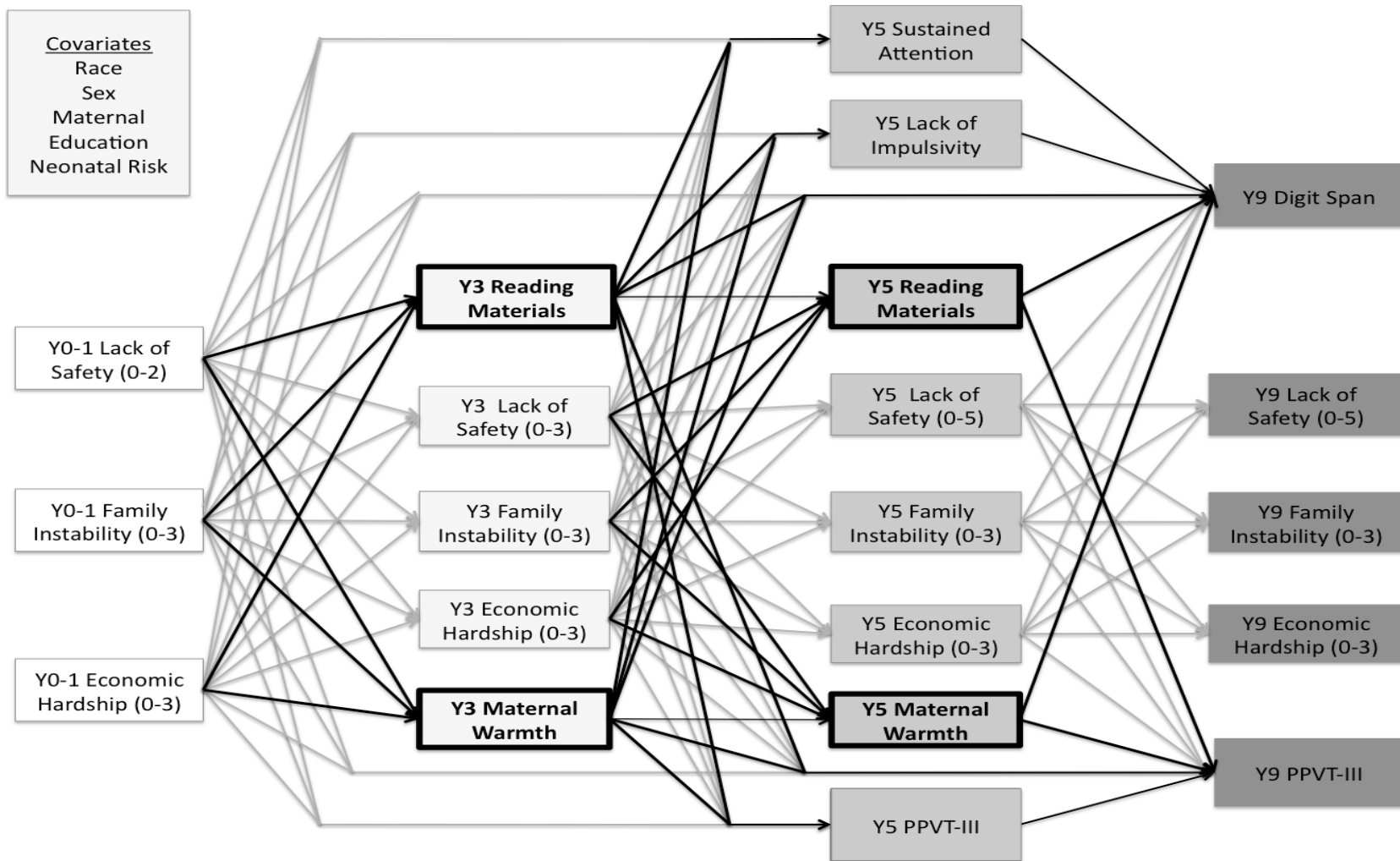
Hypotheses were analyzed using path analysis in Mplus 7.3 (62). Path analysis is a type of structural equation modeling that estimates a system of equations between observed exogenous (predicting) and endogenous (mediating or outcome) variables. Unlike latent variable models, path models assume no measurement error in observed variables. Path analysis distinguishes three types of effects between variables: a direct effect is the influence that one variable has on another that is not mediated by any other variable in the model; an indirect effect is the influence of one variable on another through mediation of at least one other variable; and the total effect is the sum of the direct and indirect effects (63).

Figure 5.1 illustrates the full analytic model for the current study. Building off of an earlier study that used same sample and examined only the paths between the adversity domains and cognitive outcomes (illustrated by the grey lines and boxes in Figure 5.1), the current study examined whether access to reading and maternal warmth mediated the relations between the adversity domains and the cognitive outcomes

(indicated by the black lines and bold black boxes in Figure 5.1). Mediation was tested by examining the joint significance of paths leading to and from the intervening variable of interest (64). We examined the significance of the indirect paths from the adversity domains in infancy to the age five and nine cognitive outcomes, through availability of reading materials and maternal warmth at age three. We also examined the significance of the indirect paths from the age three adversity domains to the age nine cognitive outcomes through availability of reading materials and maternal warmth at age five. According to MacKinnon and colleagues, for mediation to be present, both paths from the domain to the mediating variable and from the mediating variable to the cognitive outcome had to be significant (64,65). Mediation was tested using the Delta Method, which, similar to the Sobel Test computes a Z-score based on the product-of-coefficients of the indirect effects (65).

We then tested for gender moderation of these indirect paths using multi-group analysis. A fully constrained model where all paths were constrained to be equal for girls and boys was compared to a model where all paths were estimated freely. A significant change in chi-square between the two models was considered evidence of moderation. The significance of the indirect path estimates were then examined to identify specific differences by gender. Models were evaluated based on the model fit (CFI >0.9 indicates acceptable fit and >0.95 close fit; RMSEA <0.08 indicates acceptable fit and <0.05 close fit), significance of path estimates, and the explained variance among the endogenous variables (63,66). Z-scores ($M=0$, $SD=1$) were created for all cognitive outcomes and used in all analyses (aside from descriptive analyses) in order to facilitate comparisons across outcomes.

Figure 5.1. Analytical Model ^a



^aThe conceptual model allowed for direct longitudinal paths between all adversity domains (paths representing one lag shown with the grey arrows; paths greater than one lag are not shown). All indicators within the same wave were correlated (not depicted in the diagram). The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

RESULTS

Preliminary Analyses

Means and standard deviations for continuous variables, and frequencies for categorical or dichotomous variables are presented in Table 5.1. Mean scores on all of the age five and age nine child cognitive assessments were at or just below the normed average. Mean scores on the adversity domains ranged from 0.2-1.3 (SD=0.2-1.4) across waves for the lack of safety domain, 0.3-0.6 (SD=0.3-0.5) for the instability domain, and 0.6-0.7 (SD=0.5-0.6) for the poverty domain. Mean scores for both the availability of reading materials and maternal warmth variables were skewed towards more favorable response.

Correlations among all model variables are displayed in Table 5.2. Correlations were classified as low (<0.1), modest (0.1-0.3), moderate (0.31-0.5) and strong (>0.5). All adversity domains were negatively correlated with all cognitive outcomes and the mediating variables, as expected. Positive correlations among adversity domains within waves were modest to moderate, all in the expected direction. Across waves, similar adversity domains were modestly to moderately correlated, all in the expected direction, indicating only moderate stability in the domains of adversity exposure over time.

We also conducted univariate path models with each cognitive outcome regressed on each of the adversity domains and mediating variables to examine the nature of these relations. For the adversity domains, higher economic hardship scores at each age significantly predicted lower scores for all cognitive outcomes. The lack of safety domain also negatively predicted each cognitive outcome, though the coefficient was not significant for the relation between lack of safety in infancy and lack of impulsivity as

well as lack of safety at age three and sustained attention. The instability domain only significantly predicted lower PPVT-III scores at ages five and nine. There were no significant effects of the instability domain on any of the attention measures. Availability of reading materials and maternal warmth at ages five and nine were significantly associated with all cognitive outcomes in the expected direction, with the exception that maternal warmth at age three was not significantly associated with lack of impulsivity at age five.

Adversity Domains and Cognitive Outcomes

We examined the direct effects of the adversity domains on all cognitive outcomes prior to adding the access to reading and maternal warmth variables in the model (this model is represented by the grey arrows in Figure 5.1). These findings are described in more detail in Figure 4.6 in Chapter 4, but serve as a starting point for the current analysis. This model fit these data well (RMSEA=0.04; CFI=0.99). After controlling for covariates and all adversity domains at each wave, significant direct effects were observed between adversity domains in infancy and age three and the cognitive outcomes. Specifically, during infancy, the lack of safety domain directly predicted PPVT-III at ages five ($\beta=-0.05$; $p<0.05$) and nine ($\beta=0.03$; $p<0.05$; unexpected direction); instability directly predicted PPVT-III at age nine ($\beta=-0.04$; $p<0.05$); and economic hardship predicted sustained attention ($\beta=-0.08$; $p<0.01$) and PPVT-III ($\beta=-0.08$; $p<0.01$) at age five. At age three, economic hardship also directly predicted age five PPVT-III ($\beta=-0.12$; $p<0.001$), age nine PPVT-III ($\beta=-0.04$; $p<0.05$), and age nine digit span ($\beta=-0.11$; $p<0.001$).

Mediation of Maternal Warmth and Reading Materials

To examine whether availability of reading materials and maternal warmth mediated the relations between adversity domains and cognitive outcomes, we next added the availability of reading materials and maternal warmth variables at ages three and five to the model (as depicted in the full model in Figure 5.1). The final model fit the data well (RMSEA=0.04; CFI=0.98; the path diagram for this model is shown in the Appendix, Figure A5.1). Nearly all significant direct paths between the adversity domains and cognitive outcomes remained significant after including the availability of reading materials and maternal warmth variables in the model, with the exception that the age three economic hardship domain no longer directly predicted age nine PPVT-III. Only the economic hardship domain directly predicted the mediating variables. Economic hardship in infancy directly predicted age three availability of reading materials ($\beta=-0.13$; $p<0.001$) and maternal warmth ($\beta=-0.07$; $p<0.05$), and economic hardship at age three directly predicted age five availability of reading materials ($\beta=-0.11$; $p<0.001$) and maternal warmth ($\beta=-0.07$; $p<0.01$). Availability of reading materials at age three directly predicted all of the cognitive outcomes, except for age five lack of impulsivity. Maternal warmth at age three only predicted the age five sustained attention and PPVT-III outcomes. There were no significant direct effects from the age five mediating variables to age nine cognitive outcomes.

We then tested the significance of specific indirect paths in cases where the direct path from the economic hardship domain to one of the mediating variables was significant, and where the direct path from the mediating variable to one of the cognitive outcomes was also significant (results shown in Table 5.3).

Availability of reading materials at age three partially mediated the relation between economic hardship in infancy and age five sustained attention ($\beta=-0.02$; $p<0.001$), age nine digit span ($\beta=-0.01$; $p<0.05$), and age nine digit span through age five sustained attention (double mediation; $\beta=-0.002$; $p<0.01$). Availability of reading materials at age three also partially mediated the relation between economic hardship in infancy and age PPVT-III ($\beta=-0.02$; $p<0.001$), age nine PPVT-III ($\beta=-0.01$; $p<0.001$), and age PPVT-III through age five PPVT-III (double mediation; $\beta=-0.01$; $p<0.001$). Age three maternal warmth also partially mediated the effect of economic hardship in infancy on age five sustained attention ($\beta=-0.02$; $p<0.001$), age five PPVT-III ($\beta=-0.01$; $p<0.05$), and age nine PPVT-II through age five PPVT-III (double mediation; $\beta=-0.003$; $p<0.05$).

The variance explained in the cognitive outcomes changed minimally with the addition of the mediating variables to the full mediation model. The mediating variables explained an additional 2% of the variance for the sustained attention outcome, an additional 3% of the variance for age five PPVT-III, an additional 1% for the age nine PPVT-III. There was no change in the variance explained for the age five lack of impulsivity and the age nine digit span outcomes with the addition of the mediating variables.

Moderation by Gender

To examine whether gender moderated this mediation process, we stratified the model for boys and girls. A significant difference in chi square results ($\chi^2=286.4$, $df=224$, $p<0.001$) comparing the stratified model where paths were constrained to be equal across genders to an unconstrained model where paths were estimated freely across genders

indicated a difference for boys and girls. We then examined differences between genders in the specific indirect effects from the adversity domains through the mediating variables to the cognitive outcomes. Again, specific indirect effects were tested only when both direct paths to and from the mediating variable were significant. Results are shown in Table 5.3 (paths are also illustrated in the Appendix, Figure A5.2). For both boys and girls, availability of reading materials at age three was the only variable to have any mediating effect in the relation between the adversity domains and the cognitive outcomes. For boys only, availability of reading materials mediated the relation between the lack of safety domain at infancy and age five PPVT-III ($\beta=-0.01$; $p<0.05$) and age nine PPVT-III through age five PPVT-III (double mediation; $\beta=-0.01$; $p<0.05$), and between family instability in infancy and age five PPVT-III ($\beta=-0.01$; $p<0.05$), and age nine PPVT-III through age five PPVT-III (double mediation; $\beta=-0.01$; $p<0.05$). For both boys and girls, availability of reading materials also mediated the relation between economic hardship in infancy and age five sustained attention, age nine digit span (though for boys this effect went through sustained attention at age five), age five PPVT-III, and age nine PPVT-III (for boys, this effect was only observed through PPVT-III at age five).

DISCUSSION

The current study examined whether two characteristics of a child's home environment – availability of reading materials and maternal warmth – mediated the relations between three adversity domains (economic hardship, family instability, and lack of safety) and child cognitive outcomes among a cohort of relatively disadvantaged urban children. In the full sample, availability of reading materials at age three partially

mediated the relation between economic hardship in infancy and sustained attention at age five, verbal ability (PPVT-III) at ages five and nine, and working memory/attention (digit span) at age nine. Maternal warmth at age three also partially mediated the relation between economic hardship in infancy and sustained attention at age five and verbal ability at ages five and nine. All mediation effects reduced the negative impact of these adversities. Mediation pathways differed by gender. For both boys and girls, availability of reading materials at age three mediated the relation between economic hardship in infancy and sustained attention at age five, verbal ability at ages five and nine, and working memory/attention at age nine. However, for boys only, availability of reading materials at age three also mediated the relation between the lack of safety and family instability domains in infancy and verbal ability at ages five and nine. We did not find any significant relationships between the adversity domains and the lack of impulsivity outcome. The lack of findings for this outcome is discussed in more detail in Chapter 4.

Mediating Role of Reading Materials

Given the ample evidence that one mechanism by which poverty negatively affects cognitive development is through the lack of cognitive stimulation that some poor children receive (20-22), we hypothesized that the availability of reading materials, an indicator of stimulation, would mediate the relation between economic hardship and all cognitive outcomes. Our findings supported this hypothesis for the outcomes of sustained attention, working memory/attention, and verbal ability.

We did not make any hypotheses about the mediating role of availability of reading materials on the relations between the lack of safety and family instability domains with

the cognitive outcomes. Although both of these domains directly predicted lower verbal ability in our full sample, neither domain predicted the availability of reading materials, and therefore, no mediating effects were observed. The availability of reading materials is dependent upon financial resources, and therefore, it makes sense that only the economic hardship domain would directly predict this variable. However, not all stimulating activities rely on financial resources. The lack of safety and family instability domains may be more likely to affect other types of cognitive stimulation that involve parent-child interactions (i.e., engaging children in games or activities, conversing or reading to children). Future studies should examine whether the lack of safety and family instability domains predict other measures of cognitive stimulation that involve parent-child interaction, and whether this type of interaction would then predict cognitive development.

Mediating Effect of Maternal Warmth

We expected that maternal warmth would mediate the effect of all three adversity domains on all cognitive outcomes because adversities are both stressful for children and can interfere with the ability of parents to provide the warmth and nurturing known to help children regulate their response to stressful situations (27,35,38). However, only the economic hardship domain negatively predicted maternal warmth in the full sample. Maternal warmth mediated the relation between economic hardship in infancy and sustained attention and verbal ability at age five. Less responsive parenting is a well established mediator of the relationship between economic hardship and cognitive

development, which is further supported by our findings (22,23). There were no direct effects between lack of safety and family instability domains and maternal warmth.

Although others have found that maternal warmth mediates the relation between a cumulative adversity index and a composite measure of attention and behavioral regulation among three year olds, five of the seven items used in the cumulative index were measures of economic hardship (24). In our previous study comparing the effects of a cumulative index to adversity domains, we found that poverty accounted for nearly all of the variance in our outcomes explained by the cumulative index (Chapter 4). Therefore, it is likely that the study above supports our current findings. Only one other study that we are aware of has examined the relation between two adversity domains, poverty and a cumulative index (consisting of low education, single parent, adolescent parent, residential instability, divorce, household density, negative events, and depression), on several parenting behaviors, and it found that only income predicted less maternal warmth whereas the cumulative index predicted other parenting behaviors, particularly negative parenting and scaffolding (26). In this same study, negative parenting and scaffolding both mediated the relation between the cumulative index and measures of executive function among pre-school age children. Together these findings suggest that examining the effects different types of adversities separately provides a more nuanced understanding of underlying pathways. Additionally, future studies should not only examine multiple adversity domains, but also more nuanced parenting behaviors.

Gender Differences

The availability of reading materials mediated the relation between economic hardship and cognitive outcomes for both boys and girls. However, only for boys did the availability of reading materials also mediate the relation between both the lack of safety and family instability domains in infancy and verbal ability at age five. In early childhood, girls develop expressive and receptive language earlier than boys (40). Additionally, mothers tend to talk more during interactions with their daughters than their sons (67,68). Later language development in boys combined with less verbal interaction may, therefore, place more importance on reading for their language development.

There is some evidence that boys' educational achievement may be more susceptible than that of girls to disadvantaged home environments (69). Although few studies have examined the mechanisms by which adversity may affect parenting of boys and girls differently, one theory suggests that boys are more susceptible to changes in parent/child interactions that occur when parents are stressed, whereas girls are more susceptible to the stress itself (70). In the current study, the availability of reading materials did not measure parent/child interaction per se, but it could indicate that boys are more susceptible to deficits in the home environment that result from adversity than are girls.

Exposures in Early Childhood

Another important finding in this study was that all significant mediating effects were only observed in early childhood. Specifically, the availability of reading materials and maternal warmth at age three mediated the relation between adversity domains in infancy and later cognitive outcomes at ages five and nine. Although economic hardship

at age three predicted the availability of reading materials and maternal warmth at age five, these variables did not predict later cognitive outcomes at age nine. These findings add to a growing body of research highlighting the importance of early parenting for children's cognitive development (19).

Limitations

There are a few caveats of this study worth noting. First, the magnitudes of significant associations between study variables were modest, though comparable to other studies multiple adversities and cognitive development with a similar number of model variables (24). Worth noting, the coefficients in these path models are partial coefficients, and represent the relations between variables, after controlling for all other associations in the model. Second, the measures of adversity used in this study were obtained from the biological mother. Due to socially desirable responses, particularly with respect to topics such as child maltreatment or domestic violence, these data may underestimate the actual occurrence of adversities and therefore attenuate their relationship to cognitive outcomes. Third, the Leiter sustained attention and lack of impulsivity measures assessed at age five measure different aspects of attention from the WISC-IV digit span assessed at age nine, limiting our confidence that the same processes were measured longitudinally. Moreover, each of these complex processes is assessed with only one instrument. Fourth, we hypothesized that adverse experiences would influence measures of attention via their impact on the stress response system and child attachment. However, we had no measures of either stress or attachment in this study. Further research would benefit by examining the relation between adversity domains and

biomarkers of stress reactivity and attachment style. Finally, while the longitudinal nature of these data is strength of the study, the timing of these assessments also presents a study limitation. The exact ages of children varied for each wave. Therefore, the study conclusions about temporality can only be generalized to early, middle, and late childhood rather than specific ages.

Implications and Conclusion

Our findings have several important implications. First, this study adds to a strong body of evidence that the lack of stimulating environments for poor children is detrimental to their cognitive development, and it lends support for early childhood interventions such as home visiting programs (71) that promote maternal warmth and responsiveness and early childhood education interventions (72) that aim to foster cognitively enriching environments in the first few years of a child's life. Although this study focused on the stimulating benefits of the availability of reading materials, evidence suggests parents do more than spend money on stimulating materials – they also promote development with the time spent engaging with their children in enriching activities (73). Second, interventions may require different approaches by gender. More research is needed to better understand how the home environment differs for boys and girls in the context of adversity, and how these differences affect development. Third, future research on multiple adverse experiences would benefit from more nuanced conceptual models that account for multiple domains of adverse exposures as well as multiple characteristics of the home environment and parent/child interactions that explain the pathways between adversities and developmental outcomes.

In conclusion, this study is the first to evaluate whether characteristics of the home environment mediate the relation between three different domains of adverse experiences on child cognitive outcomes. We add to the body of evidence that the inverse relationship between economic hardship and cognitive development in early childhood is mediated by deficits in materials that support early literacy and language stimulation. The mediating role of maternal warmth in the relation between economic hardship and sustained attention provides some support for the neurobiological influence of adverse experiences on the development of executive function. Receptive language development in boys may be particularly sensitive to the availability of stimulating activities in unsafe or unstable home environments. These findings lend support for early interventions to improve parenting and the home environment and enriching activities for children exposed to adversity in early life.

Table 5.1. Description of Key Variables (N=2976)

	Final Analytic Sample			
	N	%	% Missing	
Mothers with \leq High School Education at Child's Birth	1087	36.6	0.1	
Mother Race			0.2	
<i>White, non-Hispanic</i>	591	19.9		
<i>Black, non-Hispanic</i>	1494	50.3		
<i>Hispanic</i>	784	26.4		
<i>Other</i>	100	3.4		
Female Child	1428	48.0	0	
Neonatal Risk	317	10.7	0.7	
	Mean	SD	% Missing	Range
Mean Maternal Age	25.1	6.0	<0.1	15-43
Year 5 Cognitive Outcomes				
<i>Sustained Attention</i>	12.9	3.3	44.3	1-19
<i>Impulse Control</i>	10.1	2.9	44.3	1-17
<i>PPVT</i>	94.3	15.4	38.1	40-139
Year 9 Cognitive Outcomes				
<i>Digit Span</i>	9.4	2.8	0.2	1-19
<i>PPVT</i>	93.1	14.9	0.5	53-159
Year 1 Lack of Safety	0.2	0.4	7.3	0-2
Year 1 Instability	0.6	0.7	9.8	0-3
Year 1 Economic Hardship	0.6	0.7	5.8	0-3
Year 3 Lack of Safety	1.1	1.1	25.1	0-5
Year 3 Instability	0.4	0.6	11.5	0-3
Year 3 Economic Hardship	0.6	0.7	25.1	0-3
Year 5 Lack of Safety	1.2	1.1	27.8	0-5
Year 5 Instability	0.3	0.5	15.5	0-3
Year 5 Economic Hardship	0.7	0.8	5.9	0-3
Year 9 Lack of Safety	1.4	1.2	9.2	0-5
Year 9 Instability	0.3	1.1	7.9	0-3
Year 9 Economic Hardship	0.7	0.8	1.5	0-3
Year 3 Parental Warmth	4.4	1.0	48.6	0-5
Year 3 Access to Reading	5.7	1.2	24.2	1-7
Year 5 Parental Warmth	4.0	1.2	45.1	0-5
Year 5 Access to Reading	7.1	1.2	26.2	2-8

Table 5.2. Correlations Among All Variables

	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	(13)	(14)	(15)	(16)	(17)	(18)	(19)	(20)	(21)	(22)	(23)	(24)	(25)	(26)	(27)	(28)
(1) Y9 Digit Span	-																											
(2) Y5 Sust. Attn.	.17	-																										
(3) Y5 Lack of Imp.	.08	.15	-																									
(4) Y9 PPVT	.34	.24	.13	-																								
(5) Y5 PPVT	.25	.34	.16	.65	-																							
(6) White	.09	.08	.06	.35	.35	-																						
(7) Black	-.01	-.18	-.11	-.29	-.20	-	-																					
(8) Hispanic	-.12	.11	.05	-.13	-.22	-	-	-																				
(9) Other	.08	.07	.09	.22	.19	-	-	-	-																			
(10) Sex	.03	.22	.14	-.04	.11	-	-	-	-	-																		
(11) > HS Edu.	.20	.16	.11	.45	.44	.28	-.14	-.25	.19	.00	-																	
(12) Neonatal Risk	-.08	-.12	-.03	-.12	-.08	-.08	.26	-.21	-.17	.05	-.05	-																
(13) Y3 Warmth	.08	.12	.05	.22	.20	.41	-.22	.04	.07	.06	.27	-.04	-															
(14) Y3 Reading	.17	.17	.05	.37	.37	.43	-.07	-.29	.14	.04	.54	.04	.22	-														
(15) Y5 Warmth	.06	.19	.08	.21	.26	.28	-.20	.05	-.03	.09	.20	-.05	.18	.17	-													
(16) Y5 Reading	.10	.10	.06	.22	.28	.34	.00	-.26	.10	.03	.39	-.01	.11	.39	.13	-												
(17) Y1 Unsafe	-.05	-.05	-.04	-.10	-.13	-.10	.09	.04	-.17	.00	-.22	.03	-.08	-.13	-.06	-.11	-											
(18) Y1 Instability	-.04	-.05	-.04	-.18	-.11	-.20	.36	-.18	-.16	-.01	-.23	.07	-.11	-.14	-.13	-.07	.12	-										
(19) Y1 Hardship	-.11	-.12	-.07	-.24	-.24	-.18	.17	.05	-.13	-.01	-.38	.08	-.14	-.26	-.11	-.16	.23	.26	-									
(20) Y3 Unsafe	-.05	-.04	-.06	-.17	-.16	-.19	.31	-.15	-.03	-.09	-.26	.05	-.17	-.18	-.11	-.11	.18	.18	.22	-								
(21) Y3 Instability	-.03	-.03	-.04	-.08	-.06	-.08	.17	-.09	-.09	.01	-.14	.04	-.06	-.06	-.06	-.04	.14	.10	.18	.20	-							
(22) Y3 Hardship	-.17	-.11	-.08	-.29	-.26	-.22	.21	.04	-.10	-.01	-.43	.11	-.18	-.28	-.15	-.22	.19	.25	.44	.26	.38	-						
(23) Y5 Unsafe	-.04	-.10	-.04	-.16	-.15	-.22	.31	-.11	-.06	-.06	-.22	.05	-.14	-.15	-.13	-.14	.19	.21	.21	.46	.22	.25	-					
(24) Y5 Instability	-.03	-.01	-.06	-.09	-.07	-.08	.15	-.06	-.15	-.01	-.09	.01	-.02	-.05	-.08	-.03	.05	.19	.14	.16	.11	.13	.20	-				
(25) Y5 Hardship	-.09	-.12	-.09	-.25	-.26	-.20	.22	-.14	-.04	.01	-.35	.10	-.17	-.21	-.15	-.17	.18	.25	.29	.21	.22	.48	.24	.29	-			
(26) Y9 Unsafe	-.07	-.04	-.07	-.11	-.10	-.17	.23	-.06	-.04	-.07	-.16	.02	-.12	-.11	-.08	-.10	.15	.15	.19	.38	.19	.17	.45	.23	.20	-		
(27) Y9 Instability	-.02	-.02	-.04	-.07	-.01	-.04	.21	-.07	-.06	.01	-.16	.05	-.01	-.04	-.07	-.03	.12	.12	.13	.11	.11	.12	.14	.15	.14	.15	-	
(28) Y9 Hardship	-.10	-.08	-.07	-.22	-.16	-.16	.20	-.02	-.17	.01	-.34	.07	-.12	-.19	-.13	-.14	.14	.14	.33	.22	.18	.40	.22	.18	.45	.24	.21	-

Z-scores used for all cognitive outcomes.

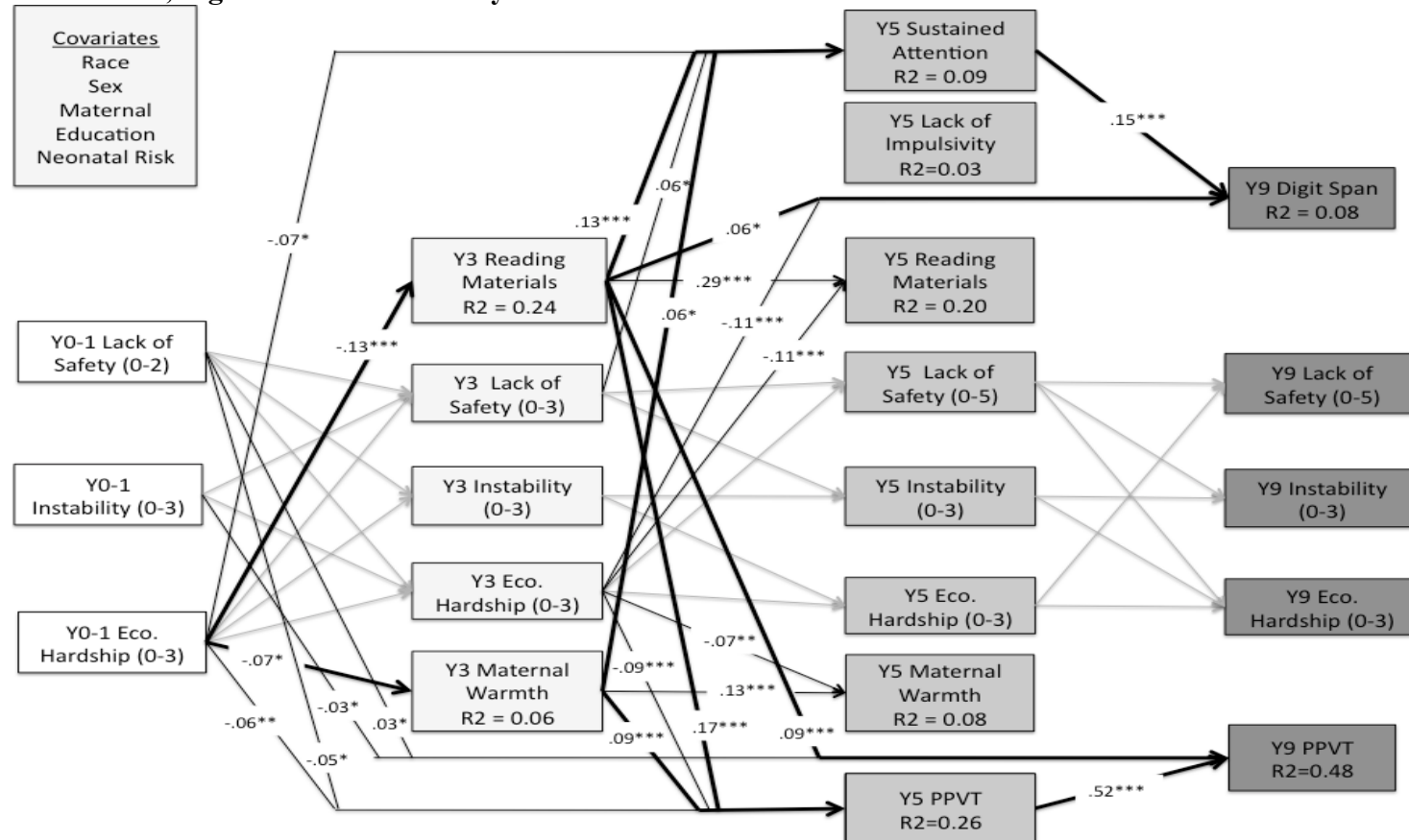
Table 5.3. Specific Indirect Effects of Adversity Domains in Infancy on Cognitive Outcomes through Age Three Reading and Maternal Warmth ^a

	Full Model	Males	Females
	Standardized β (SE)	Standardized β (SE)	Standardized β (SE)
<i>Y1 Lack of Safety to Y5 & Y9 Outcomes</i>			
Y1 Lack of Safety → Y3 Reading	-- --	-0.056 (0.027)*	-- --
→ Y5 Sustained Attention	-- --	-- --	-- --
→ Y5 Lack of Impulsivity	-- --	-- --	-- --
→ Y9 Digit Span	-- --	-- --	-- --
→ Y5 PPVT-III	-- --	-0.011 (0.006)*	-- --
→ Y9 PPVT-III	-- --	-- --	-- --
→ Y5 PPVT-III → Y9 PPVT-III	-- --	-0.006 (0.003)*	-- --
Y1 Lack of Safety → Y3 Warmth	-- --	-- --	-- --
<i>Y1 Family Instability to Y5 & Y9 Outcomes</i>			
Y1 Instability → Y3 Reading	-- --	-0.059 (0.028)*	-- --
→ Y5 Sustained Attention	-- --	-- --	-- --
→ Y5 Lack of Impulsivity	-- --	-- --	-- --
→ Y9 Digit Span	-- --	-- --	-- --
→ Y5 PPVT-III	-- --	-0.012 (0.006)*	-- --
→ Y9 PPVT-III	-- --	-- --	-- --
→ Y5 PPVT-III → Y9 PPVT-III	-- --	-0.006 (0.003)*	-- --
Y1 Instability → Y3 Warmth	-- --	-- --	-- --
<i>Y1 Economic Hardship to Y5 & Y9 Outcomes</i>			
Y1 Hardship → Y3 Reading	-0.128 (0.020)**	-0.078 (0.028)**	-0.189 (0.029)***
→ Y5 Sustained Attention	-0.016 (0.005)***	-0.013 (0.006)*	-0.017 (0.008)*
→ Y5 Lack of Impulsivity	-- --	-- --	-- --
→ Y9 Digit Span	-0.008 (0.003)*	-- --	-0.019 (0.008)*
→ Y5 Sust. Attn. → Y9 Digit Span	-0.002 (0.001)**	-0.002 (0.001)*	-- --
→ Y5 PPVT-III	-0.022 (0.005)***	-0.016 (0.006)*	-0.023 (0.007)**
→ Y9 PPVT-III	-0.012 (0.003)***	-- --	-0.026 (0.007)***
→ Y5 PPVT-III → Y9 PPVT-III	-0.011 (0.002)***	-0.008 (0.003)*	-0.012 (0.004)**
Y1 Hardship → Y3 Warmth	-0.068 (0.027)*	-- --	-0.100 (0.038)**
→ Y5 Sustained Attention	-0.016 (0.005)***	-- --	-- --
→ Y5 Lack of Impulsivity	-- --	-- --	-- --
→ Y9 Digit Span	-- --	-- --	-- --
→ Y5 PPVT-III	-0.006 (0.003)*	-- --	-- --
→ Y9 PPVT-III	-- --	-- --	-- --
→ Y5 PPVT-III → Y9 PPVT-III	-0.003 (0.002)*	-- --	-- --

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$; Y1 = year one; Y3 = year three; Y5 = year five; Y9 = year nine; Sust. Attn. = Sustained Attention. Only significant paths are shown.

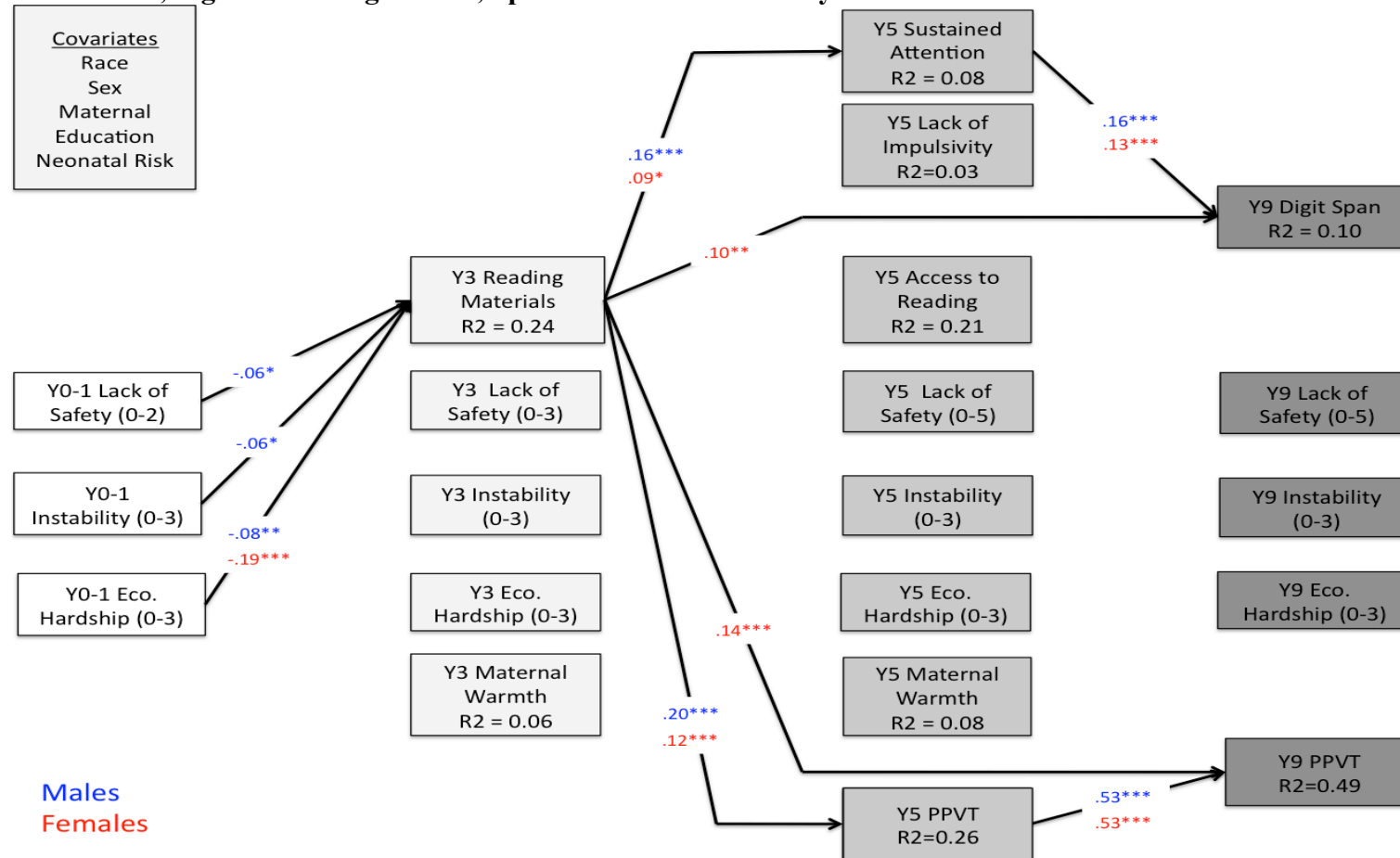
^a The headings in the grey rows draw attention to a specific domain from year one and its relation to the cognitive outcomes. The first direct path listed under the heading represents the direct effect from the year one domain to a year three mediating variable. If this direct path was significant, then the subsequent indirect effects (from the year one domain to the year five and year nine outcomes through that year three mediating variable) were then listed below.

APPENDIX, Figure A5.1. Final Analytic Model ^a



^a * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$; Significant paths shown. Bolded paths represent significant indirect pathways. Standardized coefficients presented. Indicators within the same wave were correlated in the expected direction (not depicted in the diagram). Cognitive outcomes within the same wave were significantly positively correlated. At age five, the lack of safety and economic hardship domains were negatively correlated with sustained attention ($r = -0.06^*$ for both), and economic hardship was negatively correlated with the PPVT-III ($r = -0.11^{***}$). At age nine, only the economic hardship domain was negatively correlated with the PPVT-III ($r = -0.06^*$). Direct, significant paths between domains with greater than one lag are also not shown; while these paths were all in the expected direction, not all were significant. The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

APPENDIX, Figure A5.2. Significant, Specific Indirect Effects by Gender ^a



^a Only significant indirect paths from the adversity domains to the cognitive outcomes are displayed. Standardized coefficients presented. Indicators within the same wave were correlated in the expected direction (not depicted in the diagram). Cognitive outcomes within the same wave were significantly positively correlated. Direct paths between the adversity domains and the cognitive outcomes are not shown. Direct, significant paths between domains with greater than one lag are also not shown; while these paths were all in the expected direction, not all were significant. The full model controlled for race, sex, maternal education and neonatal risk; each covariate was correlated with one another and pointed to all other variables in the model.

REFERENCES

1. Felitti VJ, Anda RF, Nordenberg D, et al. Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine* 1998;14:245–58.
2. Dong M, Anda RF, Felitti VJ, et al. The interrelatedness of multiple forms of childhood abuse, neglect, and household dysfunction. *Child Abuse & Neglect* 2004;28:771–84.
3. Evans GW, Li D, Whipple SS. Cumulative risk and child development. *Psychological Bulletin* 2013;139:1342.
4. Klebanov PK, Brooks-Gunn J. Cumulative, Human Capital, and Psychological Risk in the Context of Early Intervention: Links with IQ at Ages 3, 5, and 8. *Annals of the New York Academy of Sciences* 2006;1094:63–82.
5. Razza RA, Martin A, Brooks-Gunn J. The implications of early attentional regulation for school success among low-income children. *Journal of Applied Developmental Psychology* 2012;33:311–9.
6. Nisbett RE, Aronson J, Blair C, et al. Intelligence: New findings and theoretical developments. *American Psychologist* 2012;67:130–59.
7. Blair C, Granger D, Peters Razza R. Cortisol reactivity is positively related to executive function in preschool children attending Head Start. *Child Development* 2005;76:554–67.
8. Blair C. How similar are fluid cognition and general intelligence? A developmental neuroscience perspective on fluid cognition as an aspect of human cognitive ability. *Behavioral and Brain Sciences* 2006;29:109–60.
9. Noble KG, Tottenham N, Casey BJ. Neuroscience perspectives on disparities in school readiness and cognitive achievement. *The Future of Children* 2005;15:71–89.
10. Nelson CA, de Hann M, Thomas KM. *Neuroscience of Cognitive Development: The Role of Experience and the Developing Brain*. Hoboken, New Jersey: John Wiley & Sons Inc; 2006.
11. Schoon I, Jones E, Cheng H, Maughan B. Family hardship, family instability, and cognitive development. *Journal of Epidemiology and Community Health* 2012;66:716–22.
12. Brown ED, Ackerman BP, Moore CA. Family adversity and inhibitory control for economically disadvantaged children: Preschool relations and associations with school readiness. *Journal of Family Psychology* 2013;27:443–52.

13. Bronfenbrenner U, Morris PA. The ecology of developmental processes. In: Lerner RM, editor. *Theoretical Models of Human Development. Handbook of Child Psychology*. New York: John Wiley & Sons Inc; 1998. p. 993–1028.
14. Flouri E, Kallis C. Adverse Life Events and Psychopathology and Prosocial Behavior in Late Adolescence: Testing the Timing, Specificity, Accumulation, Gradient, and Moderation of Contextual Risk. *Journal of the American Academy of Child & Adolescent Psychiatry* 2007;46:1651–9.
15. Ayoub C, O'Connor E, Rappolt-Schlichtmann G, Vallotton C, Raikes H, Chazan-Cohen R. Cognitive skill performance among young children living in poverty: Risk, change, and the promotive effects of Early Head Start. *Early Childhood Research Quarterly* 2009;24:289–305.
16. Krishnakumar A, Black MM. Longitudinal predictors of competence among African American children: The role of distal and proximal risk factors. *Journal of Applied Developmental Psychology* 2002;23:237–66.
17. Poehlmann J. Children's family environments and intellectual outcomes during maternal incarceration. *Journal of Marriage and Family* 2005;67:1275–85.
18. Burchinal M, Vernon-Feagans L, Cox M, Key Family Life Project Investigator. Cumulative Social Risk, Parenting, and Infant Development in Rural Low-Income Communities. *Parenting: Science and Practice* 2008;8:41–69.
19. Rodriguez ET, Tamis-LeMonda CS, Spellmann ME, et al. The formative role of home literacy experiences across the first three years of life in children from low-income families. *Journal of Applied Developmental Psychology* 2009;30:677–94.
20. Guo G, Harris KM. The mechanisms mediating the effects of poverty on children's intellectual development. *Demography* 2000;37:431–47.
21. Brooks-Gunn J, Duncan GJ. The effects of poverty on children. *The Future of Children* 1997;7:55–71.
22. Yeung WJ, Linver MR, Brooks-Gunn J. How money matters for young children's development: Parental investment and family processes. *Child Development* 2002;73:1861–1879.
23. Gershoff ET, Aber JL, Raver CC, Lennon MC. Income Is Not Enough: Incorporating Material Hardship Into Models of Income Associations With Parenting and Child Development. *Child Development* 2007;78:70–95.
24. Mistry RS, Benner AD, Biesanz JC, Clark SL, Howes C. Family and social risk, and parental investments during the early childhood years as predictors of low-income children's school readiness outcomes. *Early Childhood Research Quarterly* 2010;25:432–49.

25. Rhoades BL, Greenberg MT, Lanza ST, Blair C. Demographic and familial predictors of early executive function development: Contribution of a person-centered perspective. *Journal of Experimental Child Psychology* 2011;108:638–62.
26. Lengua LJ, Kiff C, Moran L, et al. Parenting Mediates the Effects of Income and Cumulative Risk on the Development of Effortful Control. *Social Development* 2013;23:631–49.
27. Evans G, Kim P, Ting A, Teshler H, Shannis D. Cumulative risk, maternal responsiveness, and allostatic load among young adolescents. *Developmental Psychology* 2007;43:341.
28. Lupien S, King S, Meaney M, McEwen B. Can poverty get under your skin? Basal cortisol levels and cognitive function in children from low and high socioeconomic status. *Development and Psychopathology* 2001;13:653–76.
29. De Bellis MD. The Psychobiology of Neglect. *Child Maltreatment* 2005;10:150–72.
30. Watts English T, Fortson BL, Gibler N, Hooper SR, De Bellis MD. The psychobiology of maltreatment in childhood. *Journal of Social Issues* 2006;62:717–36.
31. Pechtel P, Pizzagalli DA. Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology* 2010;214:55–70.
32. McEwen BS, Gianaros PJ. Central role of the brain in stress and adaptation: Links to socioeconomic status, health, and disease. *Annals of the New York Academy of Sciences* 2010;1186:190–222.
33. Blair C, Granger DA, Willoughby M, et al. Salivary Cortisol Mediates Effects of Poverty and Parenting on Executive Functions in Early Childhood. *Child Development* 2011;82:1970–84.
34. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci* 2009;10:434–45.
35. Gunnar M, Quevedo K. The Neurobiology of Stress and Development. *Annu Rev Psychol* 2007;58:145–73.
36. McEwen BS. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. *European Journal of Pharmacology* 2008;583:174–85.
37. Evans GW, Kim P. Childhood Poverty and Health: Cumulative Risk Exposure and Stress Dysregulation. *Psychological Science* 2007;18:953–7.

38. Repetti RL, Taylor SE, Seeman TE. Risky families: Family social environments and the mental and physical health of offspring. *Psychological Bulletin* 2002;128:330.
39. Pasco Fearon RM, Belsky J. Attachment and Attention: Protection in Relation to Gender and Cumulative Social -Contextual Adversity. *Child Development* 2004;75:1677–93.
40. Vallotton CD, Harewood T, Ayoub CA, Pan B, Mastergeorge AM, Brophy-Herb H. Buffering boys and boosting girls: The protective and promotive effects of Early Head Start for children's expressive language in the context of parenting stress. *Early Childhood Research Quarterly* 2012;27:695–707.
41. Reichman NE, Teitler JO, Garfinkel I, McLanahan SS. Fragile families: Sample and design. *Children and Youth Services Review* 2001;23:303–26.
42. Campbell JM, Brown RT, Cavanagh SE, Vess SF, Segall MJ. Evidence-based Assessment of Cognitive Functioning in Pediatric Psychology. *Journal of Pediatric Psychology* 2008;33:999–1014.
43. Wechsler D. Wechsler Intelligence Scale for Children: WISC-IV ®. 4 ed. San Antonio, TX: Harcourt Assessment; 2003.
44. Dunn LM. Peabody Picture Vocabulary Test. 3rd ed. Circle Pines, MN: American Guidance Service; 1997.
45. Roid GH, Miller LJ. Leiter International Performance Scale-Revised. Wood Dale, IL: Stoelting Co; 1997.
46. Straus MA, Hamby SL, Finkelhor D, Moore DW, Runyan D. Identification of child maltreatment with the Parent-Child Conflict Tactics Scales: Development and psychometric data for a national sample of American parents. *Child Abuse & Neglect* 1998;22:249–70.
47. Straus MA, Field CJ. Psychological aggression by American parents: National data on prevalence, chronicity, and severity. *Journal of Marriage and Family* 2003;65:795–808.
48. Straus MA, Stewart JH. Corporal punishment by American parents: National data on prevalence, chronicity, severity, and duration, in relation to child and family characteristics. *Clin Child Fam Psychol Rev* 1999;2:55–70.
49. Sweet J, Bumpass L, Call V. The Design and Content of the National Survey of Families and Households. University of Wisconsin--Madison. Center for Demography and Ecology; 1988. Retrieved 2015 Sept 2. Available from: <http://www.ssc.wisc.edu/cde/nsfhwf/nsfh1.pdf>.
50. Lloyd S. The Effects of Violence on Women's Employment. *Law and Policy*

1997;19:139–67.

51. Waldfogel J, Craigie TA, Brooks-Gunn J. Fragile families and child wellbeing. *The Future of Children* 2010;20:87.
52. Cavanagh SE, Huston AC. Family instability and children's early problem behavior. *Social Forces* 2006;85:551–81.
53. Craigie T-AL, Brooks-Gunn J, Waldfogel J. Family structure, family stability and outcomes of five-year-old children. *Families, Relationships and Societies* 2012;1:43–61.
54. Kessler RC, Andrews G, Mroczek D, Ustun B, Wittchen HU. The World Health Organization Composite International Diagnostic Interview Short -Form (CIDI -SF). *International journal of methods in psychiatric research* 2005;7:171–85.
55. Bureau UC. Survey on Income and Program Participation. Washington, DC: 1996.
56. Schlomer GL, Bauman S, Card NA. Best practices for missing data management in counseling psychology. *Journal of Counseling Psychology* 2010;57:1–10.
57. Caldwell MB, Bradley HR. Home Observation for Measurement of the Environment: Administration Manual. Tempe, AZ: Family & Human Dynamics Research Institute, Arizona State University; 2003.
58. Leventhal T, Selner-O'Hagan MB, Brooks-Gunn J, Bingenheimer JB, Earls FJ. The Homelife Interview from the Project on Human Development in Chicago Neighborhoods: Assessment of parenting and home environment for 3-to 15-year-olds. *Parenting: Science and Practice* 2004;4:211–41.
59. Wechsler D. Wechsler Adult Intelligence Scale - revised (WAIS-R Manual). Harcourt Brace Jovanovich; 1981.
60. Little TD, Jorgensen TD, Lang KM, Moore EWG. On the Joys of Missing Data. *Journal of Pediatric Psychology* 2014;39:151–62.
61. Enders CK. Applied Missing Data Analysis. New York: Guilford Publications; 2010.
62. Muthén BO, Muthén LK. Mplus User's Guide. Seventh Edition. Los Angeles: 2012.
63. Bollen KA. Structural Equations with Latent Variables. Canada: John Wiley and Sons, Inc; 1989.
64. MacKinnon DP, Lockwood CM, Hoffman JM. A comparison of methods to test mediation and other intervening variable effects. *Psychological Methods*

2002;7:1–35.

65. Fritz MS, MacKinnon DP. Required sample size to detect the mediated effect. *Psychological Science* 2007;18:233–9.
66. Little TD. *Longitudinal Structural Equation Modeling*. New York: The Guilford Press; 2013.
67. Barnett MA, Gustafsson H, Deng M, Mills-Koonce WR, Cox M. Bidirectional Associations Among Sensitive Parenting, Language Development, and Social Competence. *Inf Child Dev* 2012;21:374–93.
68. Clearfield MW, Nelson NM. Sex Differences in Mothers' Speech and Play Behavior with 6-, 9-, and 14-Month-Old Infants. *Sex Roles* 2006;54:127–37.
69. Mensah FK, Kiernan KE. Gender differences in educational attainment: influences of the family environment. *British Educational Research Journal* 2010;36:239–60.
70. Conger RD, Conger KJ, Elder GH Jr, Lorenz FO. Family economic stress and adjustment of early adolescent girls. *Developmental Psychology* 1993;29:206-219.
71. Paulsell D, Avellar S, Martin ES, Del Grosso P. Home visiting evidence of effectiveness review: Executive summary 2010; Retrieved 2015 Sept 8; Available from: http://homvee.acf.hhs.gov/HomVEE_Executive_Summary.pdf.
72. Camilli G, Vargas S, Ryan S, Barnett WS. Meta-analysis of the effects of early education interventions on cognitive and social development. *Teach Coll Rec* 2010; 112:579-620.
73. Kalil A. Proposal 2: Addressing the Parenting Divide to Promote Early Childhood Development for Disadvantaged Children. *Policies to Address Poverty in America* 2014; Retrieved 2015 Sept 8; Available from: http://www.hamiltonproject.org/files/downloads_and_links/parenting_divide_early_child_development_kalil.pdf.

CHAPTER 6

CONCLUSIONS AND IMPLICATIONS FOR RESEARCH, POLICY AND PRACTICE

DISSERTATION AIMS

The overarching goal for this dissertation was to examine the relationship between multiple adverse experiences and child cognitive development—a critical aspect of academic success and well-being (1). The three specific aims addressed in this dissertation responded to existing gaps in the literature outlined in the introductory chapter. First, through a systematic review of the literature, we aimed to describe what was known about the relation between multiple adverse experiences and child cognitive development and to summarize the current literature in the form of a guiding conceptual framework. Second, we examined the timing of adverse experiences and the significance of specific adversity domains in relation to cognitive development using data from the Fragile Families and Child Wellbeing (FFCW) Study. Third, we examined underlying mechanisms explaining the relations between domains of adversities and cognitive development as well as gender differences in these relations, also using data from the FFCW Study. A summary of the conclusions for each of these aims, along with implications for future research, policy and practice, is described below.

CONCLUSIONS FROM DISSERTATION AIMS

Aim 1: Systematic Review and Conceptual Framework

In Aim 1, we described what was known about multiple adverse experiences and child cognitive development through a systematic review of the literature. Prior to embarking on this literature review, it was fairly well established that children who experience a greater number of adversities are more likely to have lower scores on various measures of cognitive performance (2). However, there were several gaps in the

literature that this review aimed to address. Across studies of multiple adversities, the number and type of adversities varied greatly, and there was little consensus as to which adversities were most important to consider in both research and practice. Furthermore, although studies using a measure of total cumulative adversities made a compelling argument that an increase in the number of total adverse experiences is associated with lower scores on cognitive assessments, the cumulative adversity approach failed to provide a theoretical foundation that could inform the choice of targets for intervention. Lacking this theoretical foundation, the mechanisms explaining the relationship between cumulative adversities and cognitive outcomes were also less understood. Additionally, little was known about the temporal influence of adverse exposures on cognitive development.

The review conducted for Aim 1 summarized findings from the literature on each of these important areas and presented a conceptual framework to guide further research and intervention. In the conceptual framework that culminated from this review, we proposed three domains of adverse experiences occurring primarily in the context of the family in early childhood, including economic hardship, family instability, and lack of safety. In the framework, we also described mechanisms by which exposure to adversity in each of these domains influences child cognitive development, namely by disrupting the safety, stability, nurturance and stimulation parents provide to their child in the early years (3). As a child's social world expands, adversities outside of the family context take on a larger role, including school-related adversities (such as bullying or school violence) or neighborhood-level adversities (such as community violence).

This review also summarized what was known about the influence of the timing of exposure to multiple adversities on child cognitive development. Although neuroscience indicates that the timing of environmental input can significantly affect developmental pathways (4-6), little is known about this topic with respect to multiple adverse exposures. Findings from this review demonstrated that both general cognitive ability and executive functioning are shaped by experience over time. However, it is difficult to disentangle the effects of sensitive periods of exposure from the effects of persistent or chronic exposures with the limited longitudinal studies that exist. There is some evidence to suggest that exposure to adversity in early childhood has lasting effects on cognitive outcomes (7,8). Additionally, the disparities in cognitive outcomes between children with high and low levels of early adverse exposures may increase as children age (9,10). Children with a greater number of adverse exposures show declines in cognitive ability over time compared to children with less adversity in their lives (9,10), and children exposed to multiple adversities show less improvement in executive function over time compared to children with less exposure (11,12).

Aim 2: Adversity Domains and Timing of Exposure

In Aim 2, we tested the conceptual framework from Aim 1 to examine whether different groupings of adverse experiences differentially predicted cognitive outcomes among a relatively disadvantaged sample of urban children in the FFCW Study. We examined two different approaches for grouping multiple adverse experiences. The first was a commonly used cumulative adversity index that summed all adverse exposures into one single score. The second approach grouped the adversities into three domains

(economic hardship, family instability, and lack of safety). We also examined the timing of adversity exposure in relation to child cognitive development for both of these approaches. Adversities were measured when children were very young (at infancy and around three years old), in the pre-school phase (five years) and late childhood (nine years), and cognitive outcomes were assessed during the preschool phase and late childhood.

When examined separately for each age, the cumulative adversity measures were inversely related to outcomes of sustained attention, working memory/attention, and verbal ability. In other words, as the number of adverse experiences increased, children were more likely to have lower scores on these cognitive assessments. When the adversity domains were examined separately for each age, only the economic hardship domain predicted these cognitive outcomes, adjusting for exposure to the other adversity domains at that age. This finding suggests that economic hardship largely drives the effect of cumulative adversities for these particular outcomes. Furthermore, adverse exposures at infancy and age three directly predicted sustained attention and verbal ability at age five, and working memory/attention and verbal ability at age nine, even after controlling for concurrent adverse exposures, confirming the hypothesis that early adverse experiences have lasting effects. After accounting for exposure to the different adversity domains at each age, economic hardship during infancy and age three still had the most salient effects on cognitive outcomes, but the lack of safety and family instability domains in infancy also directly predicted later verbal ability at ages five and nine. Despite the statistical significance of these findings, these observed relationships were only modest with respect to the magnitude of their effects.

This study made several important contributions to the literature. It was the first to examine three different domains of adversities in relation to child cognitive outcomes and to examine exposure to these different domains at multiple time points across early, middle and late childhood. Therefore, we showed the relative importance of economic hardship over other adverse exposures for cognitive development. Additionally, the lasting impact of early adverse exposures adds to a growing body of evidence for an early sensitive period for cognitive development (13,14). Although it was not the focus of this study, the additional finding that the control variables, and particularly maternal education and race, explained most of the variance in all of the cognitive outcomes was also noteworthy. These findings indicate that maternal education may be an important target for intervention, and unmeasured correlates of race are worth further investigation.

Aim 3: Mediating Role of the Home Environment

Building further on the conceptual framework proposed in Aim 1 and the findings from Aim 2, we then examined in Aim 3 whether characteristics of the home environment, particularly the availability of reading materials and maternal warmth, mediated the relation between exposure to the three domains of adversities in early childhood and later cognitive outcomes. Greater economic hardship was expected to result in fewer reading materials, thus negatively impacting cognitive outcomes due to fewer opportunities for cognitive stimulation in the home. Greater exposure to adversities in all three domains was expected to negatively influence cognitive development by limiting maternal warmth. Additionally, this aim explored whether gender moderated these effects.

As hypothesized, greater economic hardship in infancy resulted in fewer reading materials at age three, and subsequently lower scores on measures of sustained attention at age five, verbal ability at ages five and nine, and working memory/attention at age nine. Maternal warmth also partially mediated the effect between economic hardship in infancy and sustained attention at age five and verbal ability at ages five and nine, all in the expected direction. However, maternal warmth did not mediate the relation between the two other adversity domains and cognitive outcomes. The availability of reading materials at age three also mediated the relation between the lack of safety and family instability domains in infancy and verbal ability at ages five and nine for boys, but not for girls.

Overall, this study was the first to longitudinally evaluate whether characteristics of the home environment mediated the relation between three different domains of adverse experiences on child cognitive outcomes. This study supported a body of evidence that economic hardship is inversely related to cognitive development in early childhood, and this relationship is mediated by deficits in materials that support early literacy and language stimulation (15-17). The mediating role of maternal warmth in the relation between economic hardship and sustained attention provided some support for the neurobiological influence of adverse experiences on the development of executive function (18,19). However, to confirm this neurobiological mechanism, further research is needed to demonstrate that exposure to economic hardship elicits a biological stress response in children, which in turn results in poorer attention, and that maternal warmth mediates the relation between economic hardship and stress reactivity. This study was also unique in that it demonstrated that these characteristics of the home environment

mediated the relation between adversity and cognitive development when children are very young, which has implications for the timing of interventions focused on changing aspects of the home environment. Furthermore, only a small number of studies have explored gender differences in mechanisms by which adverse experiences differentially affect the home environment for boys and girls and their subsequent cognitive outcomes. The finding that verbal ability in boys may be more sensitive to the availability of stimulating activities in unsafe or unstable home environments has implications for interventions, such as home visiting programs, that focus on improving the home environment for low-income, new mothers. Strategies may differ depending upon the gender of the child.

Overall Contribution

In summary, several important contributions were made to the literature through this dissertation. Methodologically, the qualitative studies were the first to examine three different adversity domains measured longitudinally across early and middle childhood in relation to characteristics of the child's home environment and child cognitive outcomes. Therefore, we were able to conclude that economic hardship had a greater effect on child cognitive development than did lack of safety or family instability. Although the finding that economic hardship is detrimental for cognitive development is well known, the relative importance of economic hardship over these other adversity domains is a unique contribution and contrary to what we expected. Due to the longitudinal design of the quantitative analyses, we were also able to conclude that adverse exposures in early childhood (in the first three years of life) have lasting effects on cognitive development

and are mediated by disruptions in a child's early home environment. Finally, we were also able to show that boys and girls may be differentially impacted by disruptions in the home environment that result from adverse experiences.

IMPLICATIONS

These conclusions have several important implications for future research, policy and practice.

Research Implications

Utility of Adversity Domains. The conceptual framework presented in Chapter 3 and the application of this framework in the analysis of the FFCW Study cohort in Chapters 4 and 5 demonstrate the utility of using a domain-based approach to researching multiple adversities. In comparison to a cumulative adversity metric, the adversity domains explained more of the variance in child cognitive outcomes. Furthermore, the three domains were differentially related to both the cognitive outcomes assessed in this study and the proposed mediating variables. These findings indicate that unlike others who have suggested non-specific effects of different adversity groupings (20), types of adverse exposures do, in fact, matter. Future research on multiple adversities should take into consideration different types of adverse experiences, and the unique, theoretically-driven mechanisms by which different types of adverse experiences may affect developmental outcomes.

Temporal Influence of Adverse Experiences. Adverse experiences in the FFCW Study cohort were only moderately stable across early and middle childhood; a

longitudinal approach permits a more thorough examination of developmental periods during which children experience adversity influences outcomes. The study conducted in Chapter 4 showed that exposure to multiple adversities when children were very young directly predicted later cognitive outcomes, even after controlling for later adverse experiences. These findings add to a growing body of evidence for an early sensitive period for cognitive development (13,14). However, this study did not decompose adverse exposures that *only* occurred in specific developmental periods. Future studies that isolate adverse exposures to specific developmental periods may provide greater insight as to whether there are specific sensitive periods during which children are most sensitive to adverse experiences.

Adversity Domains Across Developmental Periods. All three of the domains proposed in this dissertation consisted of adversities likely to occur in the child's immediate home environment, and therefore, these domains may have more salience in early childhood when a child's social world consists primarily of the home. The majority of research studies reviewed throughout this dissertation also focused on adversities in the home environment and outcomes in early childhood. Chapters 4 and 5 of this dissertation add to just a handful of studies that examined the relations between multiple adverse experiences on cognitive outcomes that extended beyond early childhood (8,12,21).

Little is known about the influence of multiple adverse experiences on cognitive development of older children, and especially adolescents (12). As children move into adolescence, their social world expands to include exposures outside of the home environment, such as schools, neighborhoods, and peers. Additionally, the adolescent

brain undergoes a wave of plasticity associated with the maturation of the prefrontal cortex and higher-level executive functions (22). Further research is warranted on the influence of multiple adverse experiences across developmental periods on adolescent outcomes. Given that exposures in adolescence extend beyond the home environment, adversity domains will need to reflect the unique contexts to which adolescents are exposed.

Further Exploration of Mechanisms. Studies examining the underlying mechanisms explaining the relation between multiple adverse experiences and child cognitive development have primarily focused on the mediating effect of the home environment. There is consistent evidence that the relationship between economic hardship and cognitive development is mediated by the lack of stimulating home environments. However, there is less consistent evidence with respect to other parenting behaviors, including factors such as parental warmth, negativity, scaffolding and limit setting, in relation to multiple domains of adversities. A more nuanced understanding of mediating pathways and whether different domains of adversities share common mechanisms could further inform targets for intervention. Additionally, other mediating factors, including nutritional quality, environmental exposures (i.e., lead), and biomarkers (i.e., cortisol levels) are also worth exploring.

Gender Differences. Underlying pathways explaining the relation between multiple adversities and cognitive development may differ for boys and girls. Only a handful of studies have examined gender differences in the relation between multiple adversities and cognitive development, and some evidence suggests that boys may be more susceptible to the changes in parent/child interactions that result from adverse

experiences in the family (23-25). Findings from this dissertation add to this limited body of evidence. Specifically, boys may be more susceptible to changes in parenting behaviors and home environments that result from unsafe or unstable contexts. Other research has suggested that girls may be more directly affected by adversity itself rather than changes in the home environment or parenting practices that may result from adverse experiences (24). More research is needed to better understand how the home environment differs for boys and girls in the context of adversity, and how these differences affect development.

Social and Emotional Development. This dissertation focused primarily on the relation between multiple adversities and child cognitive outcomes, and the findings indicate a strong relation between economic hardship and child cognitive development. However, multiple adverse experiences also affect other aspects of social and emotional development (2). Although the family instability and lack of safety domains did not show robust effects on cognitive outcomes in this study after accounting for economic hardship, they may be more likely to influence other social-emotional or behavioral outcomes (8). Therefore, future research should examine the effect of these adversity domains on different social, emotional and behavioral outcomes.

Protective Factors, Buffers and Interventions. More research is also needed on factors that protect against or buffer the effect of adverse experiences. Among the studies that met the criteria for the literature review in Chapter 3, only a small portion aimed to identify factors that buffered or protected against multiple adversities, and these all focused on the role of early childhood education programs, such as Early Head Start, which buffered the effects of multiple adversities on child cognitive ability (8,9,26).

Resiliency research has long noted that not all young people exposed to adversity are doomed to failure (27-29). Studies are beginning to merge these fields, but more can be done in this area to explore characteristics of children, families, schools and neighborhoods that foster resiliency in the context of multiple adverse experiences (30).

Policy Implications

Economic Hardship. As public and political will increases to address adverse childhood experiences, it is important to emphasize the unique effects of specific types of adverse experiences. This dissertation showed that economic hardship had the most robust effects on children's cognitive development, even after accounting for other adverse exposures. The Centers of Disease Control and Prevention's Behavioral Risk Factor Surveillance System (BRFSS) now has standardized questions that assess adverse experiences among participating states (31). Currently these questions do not address economic hardship, and they should. Tracking adverse exposures is a powerful tool for influencing state and national agendas to respond to childhood adversities (32).

Additionally, there is enough evidence to claim that poverty negatively impacts children's cognitive development. With 22% of children in the U.S. living below the federal poverty level, policies that specifically aim to alleviate economic hardship are sorely needed. The Earned Income Tax Credit (EITC) is one positive example, which directly supplements the income of low-income workers. One of the many benefits of the EITC is improved cognitive performance for children of recipients (33).

Early Childhood Interventions. This dissertation has implications for intervention timing and lends support for programs that intervene within the first few years of a

child's life. Interventions that promote positive parenting practices in the home environment and enhance cognitive stimulation have been successful. Home visiting programs, for example, are designed to intervene with high-risk families early in a child's life, and have improved parenting practices that shape future outcomes for children (34). Additionally, early childhood education programs that aim to provide children with early experiences and stimulation are associated with better cognitive outcomes among children who have experienced adversity (8,9) and have reduced disparities in achievement evident by the time that socioeconomically disadvantaged children enter kindergarten (35). Policies that support these programs are warranted.

Maternal Education. The analyses of the FFCW Study showed children whose mothers had a high school education or less scored significantly lower on all cognitive outcomes. The association of maternal education on child cognitive outcomes and achievement is well documented (36,37). Efforts to educate girls may have intergenerational effects on their children's cognitive development. Increasing maternal education during the early years of their child's life has even been shown to improve cognitive outcomes for children (36).

Practice Implications

Clinical Practice. Access to a medical home has been shown to be protective for child well-being among children exposed to adverse experiences (38). There has been increased focus by pediatricians and public health practitioners to screen children for adverse experiences and to connect children experiencing high levels of adversity to appropriate services (30-32,39). Again, many of these screening tools do not currently

assess for economic hardship. Findings from this dissertation indicate that economic hardship is an important adversity to incorporate into such assessments. Additionally, given the significance of adverse exposures at the time of the child's birth, it is important for clinicians to screen for adversity and link new mothers to appropriate interventions and services during prenatal care.

Gender Differences in Home Environments. Although there is limited research on gender differences in response to adverse exposures, this study adds to a small body of evidence that the underlying mechanisms by which adverse experiences affects boys and girls differs. Interventions for boys may require more attention to disruptions in parent/child interactions in the context of adversity whereas girls may benefit more from interventions that reduce exposure to actual adversities (25).

CONCLUDING COMMENTS

Multiple adverse experiences are detrimental to cognitive development, and economic hardship may drive these effects. Given that different types of adverse exposures affect cognitive development differently, a domain-based approach to the study of multiple adverse experiences is warranted. Furthermore, exposures as early as infancy have lasting effects on trajectories of cognitive development with disparities in general cognitive ability and executive functions increasing over time. The relations between multiple adversities, and particularly economic hardship, and cognitive development is partially explained by disruptions in the home environment that limit parent and child interactions that foster safe, stable, nurturing, and stimulating home environments. These disruptions may impact boys and girls differently. Efforts to

promote safe, stable, nurturing and stimulating home environments through early interventions are promising strategies to improve outcomes for youth. Policies that alleviate poverty for mothers and boost maternal education may also positively impact future generations. Additionally, continued public health surveillance of adverse childhood experiences, including assessments of economic hardship, can help to build support for programs, policies and practices that both prevent adversity and foster resiliency in children, families and communities.

REFERENCES

1. Noble KG, Tottenham N, Casey BJ. Neuroscience perspectives on disparities in school readiness and cognitive achievement. *The Future of Children* 2005;15:71–89.
2. Evans GW, Li D, Whipple SS. Cumulative risk and child development. *Psychological Bulletin* 2013;139:1342.
3. Centers for Disease Control and Prevention. Essentials for Childhood: Steps to create safe, stable, nurturing relationships and environments 2014. Retrieved 2015 Sept 8. Available from: http://www.cdc.gov/violenceprevention/pdf/essentials_for_childhood_framework.pdf.
4. Lupien SJ, McEwen BS, Gunnar MR, Heim C. Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nat Rev Neurosci* 2009;10:434–45.
5. Pechtel P, Pizzagalli DA. Effects of early life stress on cognitive and affective function: an integrated review of human literature. *Psychopharmacology* 2010;214:55–70.
6. McEwen BS. Early life influences on life-long patterns of behavior and health. *Ment Retard Dev Disabil Res Rev* 2003;9:149–54.
7. Mistry RS, Benner AD, Biesanz JC, Clark SL, Howes C. Family and social risk, and parental investments during the early childhood years as predictors of low-income children's school readiness outcomes. *Early Childhood Research Quarterly* 2010;25:432–49.
8. Klebanov PK, Brooks-Gunn J. Cumulative, Human Capital, and Psychological Risk in the Context of Early Intervention: Links with IQ at Ages 3, 5, and 8. *Annals of the New York Academy of Sciences* 2006;1094:63–82.
9. Ayoub C, O'Connor E, Rappolt-Schlichtmann G, Vallotton C, Raikes H, Chazan-Cohen R. Cognitive skill performance among young children living in poverty: Risk, change, and the promotive effects of Early Head Start. *Early Childhood Research Quarterly* 2009;24:289–305.
10. Burchinal MR, Roberts JE, Hooper S, Zeisel SA. Cumulative risk and early cognitive development: A comparison of statistical risk models. *Developmental Psychology* 2000;36:793–807.
11. Brown ED, Ackerman BP, Moore CA. Family adversity and inhibitory control for economically disadvantaged children: Preschool relations and associations with school readiness. *Journal of Family Psychology* 2013;27:443–52.
12. Cybele Raver C, McCoy DC, Lowenstein AE, Pess R. Predicting individual

differences in low-income children's executive control from early to middle childhood. *Developmental Science* 2013;16:394–408.

13. Doyle O, Harmon CP, Heckman JJ, Tremblay RE. Investing in early human development: Timing and economic efficiency. *Economics & Human Biology* 2009;7:1–6.
14. Shonkoff JP, Garner AS, The Committee on Psychosocial Aspects of Child and Family Health, Committee on Early Childhood, Adoption, and Dependent Care, and Section on Developmental and Behavioral Pediatrics, et al. The Lifelong Effects of Early Childhood Adversity and Toxic Stress. *Pediatrics* 2011;129:e232–46.
15. Guo G, Harris KM. The mechanisms mediating the effects of poverty on children's intellectual development. *Demography* 2000;37:431–47.
16. Brooks-Gunn J, Duncan GJ. The effects of poverty on children. *The Future of Children* 1997;7:55–71.
17. Yeung WJ, Linver MR, Brooks-Gunn J. How money matters for young children's development: Parental investment and family processes. *Child Development* 2002;73:1861–1879.
18. Blair C, Granger DA, Willoughby M, et al. Salivary Cortisol Mediates Effects of Poverty and Parenting on Executive Functions in Early Childhood. *Child Development* 2011;82:1970–84.
19. Gunnar M, Quevedo K. The Neurobiology of Stress and Development. *Annu Rev Psychol* 2007;58:145–73.
20. Sameroff AJ, Seifer R, Barocas R, Zax M, Greenspan S. Intelligence quotient scores of 4-year-old children: social-environmental risk factors. *Pediatrics* 1987;79:343–50.
21. Sameroff AJ, Seifer R, Baldwin A, Baldwin C. Stability of intelligence from preschool to adolescence: The influence of social and family risk factors. *Child Development* 1993;64:80–97.
22. Casey BJ, Jones RM, Somerville LH. Braking and Accelerating of the Adolescent Brain. *J Res Adolesc* 2011;21:21–33.
23. Mensah FK, Kiernan KE. Gender differences in educational attainment: influences of the family environment. *British Educational Research Journal* 2010;36:239–60.
24. Conger RD, Conger KJ, Elder GH Jr, Lorenz FO. Family economic stress and adjustment of early adolescent girls. *Developmental Psychology* 1993;29:206–219.
25. Vallotton CD, Harewood T, Ayoub CA, Pan B, Mastergeorge AM, Brophy-Herb

- H. Buffering boys and boosting girls: The protective and promotive effects of Early Head Start for children's expressive language in the context of parenting stress. *Early Childhood Research Quarterly* 2012;27:695–707.
26. Liaw F-R, Brooks-Gunn J. Cumulative familial risks and low-birthweight children's cognitive and behavioral development. *Journal of Clinical Child Psychology* 1994;23:360–272.
 27. Masten AS, Powell JL. A resilience framework for research, policy, and practice. In: Luthar S, editor. *Resilience and vulnerability: Adaptation in the context of childhood adversities*. Cambridge: Cambridge University Press; 2003. p. 1–25.
 28. Masten AS, Hubbard JJ, Gest SD, Tellegen A, Garmezy N, Ramirez M. Competence in the context of adversity: Pathways to resilience and maladaptation from childhood to late adolescence. *Development and Psychopathology* 1999;11:143–69.
 29. Werner E. Risk, resilience, and recovery: Perspectives from the Kauai Longitudinal Study. *Development and Psychopathology* 1993;5:503–3.
 30. Bethell CD, Newacheck P, Hawes E, Halfon N. Adverse Childhood Experiences: Assessing The Impact On Health And School Engagement And The Mitigating Role Of Resilience. *Health Affairs* 2014;33:2106–15.
 31. Centers for Disease Control and Prevention. Adverse childhood experiences reported by adults---five states, 2009. *MMWR Morbidity and mortality weekly report* 2010;59:1609.
 32. Felitti VJ, Anda RF. The Lifelong Effects of Adverse Childhood Experiences. In: *Child Maltreatment*. Saint Louis: STM Learning, Inc; 2014:203–16.
 33. Dahl GB, Lochner L. The Impact of Family Income on Child Achievement: Evidence from the Earned Income Tax Credit. *American Economic Review* 2012;102:1927–56.
 34. Paulsell D, Avellar S, Martin ES, Del Grosso P. Home visiting evidence of effectiveness review: Executive summary 2010; Retrieved 2015 Sept 8; Available from: http://homvee.acf.hhs.gov/HomVEE_Executive_Summary.pdf.
 35. Camilli G, Vargas S, Ryan S, Barnett WS. Meta-analysis of the effects of early education interventions on cognitive and social development. *Teach Coll Rec* 2010; 112:579-620.
 36. Harding JF. Increases in maternal education and low-income children's cognitive and behavioral outcomes. *Developmental Psychology* 2015;51:583–99.
 37. Carneiro P, Meghir C, Parey M. Maternal Education, Home Environments, and the Development of Children and Adolescents. *Journal of the European Economic*

Association 2012;11:123–60.

38. Balistreri KS. Adverse Childhood Experiences, the Medical Home, and Child Well-Being. *Matern Child Health J* 2015;1–9.
39. Anda RF, Butchart A, Felitti VJ, Brown DW. Building a Framework for Global Surveillance of the Public Health Implications of Adverse Childhood Experiences. *American Journal of Preventive Medicine* 2010;39:93–8.

Stephanie Allison Guinosso, PhD, MPH

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EDUCATION

- Johns Hopkins Bloomberg School of Public Health – Baltimore, MD** 2010-present
PhD Candidate, Department of Population, Family and Reproductive Health
Dissertation: Multiple Adverse Experiences and Child Cognitive Development
- Johns Hopkins Bloomberg School of Public Health – Baltimore, MD** 2003-2004
MPH, Social and Behavioral Sciences
Capstone: Future Directions for Structural Interventions in the Prevention of HIV
- University of California, Santa Cruz – Santa Cruz, CA** 1994-1998
BA, Molecular, Cellular and Developmental Biology

AWARDS, GRANTS

- Jack Kent Cooke Dissertation Fellowship Award** 2014
- Delta Omega Alpha Scholarship Award, Johns Hopkins** 2014
- Fragile Families Summer Institute Scholarship, Columbia University** 2013
- Cheryl Alexander Memorial Fund for Adolescent Health, Johns Hopkins** 2011
- Urban Health Institute Small Grants Award, Johns Hopkins** 2011
- Highest Honors in the Major and College Honors, UC Santa Cruz** 1998

RESEARCH EXPERIENCE

- Johns Hopkins Bloomberg School of Public Health – Baltimore, MD** 2013-2014
Doctoral Research
- Conducted a systematic review of adverse childhood experiences (ACEs) and child cognitive development resulting in a conceptual framework for future research and intervention.
 - Analyzed relationship between ACEs and cognitive development (focusing on timing of exposures and mediating factors) using longitudinal data from the Fragile Families and Child Well-being study (factor analysis, regression analysis, structural equation modeling, and mediation analysis).
- Johns Hopkins Bloomberg School of Public Health – Baltimore, MD** 2011-2013
Graduate Student Researcher
- Analyzed the relationship between adolescent neurocognitive skills and driving behavior (regression analysis).

- Evaluated the impact of the Urban Health Institute's small grants program towards promoting partnerships between academic and community entities (instrument design, qualitative data collection and synthesis).
- Evaluated an STI testing initiative for community supervised, high-risk young women through a small grant with the Baltimore Department of Juvenile Services.

Education, Training and Research Associates – Oakland, CA

Research Associate

2013

- Conducted literature review on the influence of violence exposure on adolescent sexual risk-taking behavior.

Research Associate

2009-2010

- Recruited schools nationwide that offer HIV testing to youth. Developed an interview field guide and conducted qualitative interviews with these school and their partners. Summarized findings into a final resource guide for school health staff to increase HIV testing among youth.

Johns Hopkins Bloomberg School of Public Health – Baltimore, MD 2

004-2006

Research Assistant

- Conducted literature review on the influence of neighborhood social capital and collective efficacy on adolescent sexual risk taking.
- Analyzed the relationship between neighborhood characteristics, parenting and sexual behavior among inner-city adolescents.
- Conducted literature review on structural interventions for the prevention of HIV/AIDS among youth.

University of California, San Francisco – San Francisco, CA

1998-2001

Staff Research Associate

- Analyzed immune responses of HIV positive patients. Constructed and tested an HIV DNA vaccine in pre-clinical trials.

TEACHING AND TRAINING EXPERIENCE

Education, Training and Research Associates – Oakland, CA

2012-present

Professional Development Consultant

- Designed and delivered workshops, trainings and webinars on a variety of public health topics to support youth-serving professionals nationwide.
- Recent topics include: adolescent brain development and implications for public health, fostering youth resiliency, recruitment and retention of youth in school-based programs, planning for program sustainability, communicating evaluation results, and effective training and facilitation skills.

Johns Hopkins Bloomberg School of Public Health – Baltimore, MD

Teaching Assistant

Winter 2011

Child Health and Development

- Course focused on developmental theory and major determinants of health and development during the first decade of life.
- Responsibilities: facilitated small group and online discussion sessions on course readings, graded student papers and presentations

Teaching Assistant

Fall 2011

Life Course Perspective on Health

- Course framed public health issues from a multi-level, life course perspective and covered basic principles of human development, from the prenatal period through senescence, examining the ways in which health reflects developmental processes.
- Responsibilities: facilitated small group discussion sessions on course readings, graded student papers and exams, counseled students on the development of conceptual frameworks for a public health problem

Teaching Assistant

Summer 2004

Problem Solving in Public Health

- Course focused on problem solving methodology for key issues in public health.
- Responsibilities: facilitated small group discussion on problem solving strategies, graded student papers

OTHER PROFESSIONAL EXPERIENCE

Innovate Health – Baltimore, MD

2015-present

Consultant

- Developing and national research and policy agenda to address adverse childhood experiences within health systems.

Education, Training and Research Associates – Oakland, CA

2012-2014

Program Manager

- Managed \$1000K contract with RTI International to develop, coordinate and deliver training and technical assistance nationwide on a variety of topics related to program implementation and evaluation for youth-service providers through the Family and Youth Services Bureau's Personal Responsibility Education Program (PREP).

Education, Training and Research Associates – Oakland, CA

2006-2010

Project Coordinator

- Managed (\$1000K) **Survive Outside Project** funded by CDC/Division of Adolescent and School Health (DASH). Developed, coordinated and facilitating training and technical assistance for juvenile justice professionals nationwide on sexual and reproductive health programs and policies, reaching 25 different States and U.S. territories. Designed evaluation survey instruments, conducted surveys, summarized survey findings into final reports.

Johns Hopkins Bloomberg School of Public Health – Baltimore, MD

2004-2006

Technical Assistance Coordinator

- Provided training and technical assistance to community coalitions nationwide for the NIH-funded Connect to Protect Project. Wrote a resource guide on structural interventions for the prevention of HIV/AIDS among youth.

Association IDEI – Quetzaltenango, Guatemala
Volunteer

2002-2003

- Assisted in establishing a clinical HIV/AIDS laboratory. Initiated HIV testing in the Hospital Rudolfo Robles using Ortho and Abbott rapid kits. Created a volunteer program providing educational activities for hospitalized tuberculosis patients.

PUBLICATIONS*

Peer-Reviewed Publications:

- Guinosso, SA, Johnson SB, Riley AW. (2015.) Multiple Adverse Experiences and Child Cognitive Development. *Pediatric Research (in press)*.
- Anderson, P, Guinosso, SA, Wilson, S, Denner, J, Coyle K. (2014). Exposure to violence and sexual risk among early adolescents in school. *Journal of Early Adolescence (in press)*.
- Kerrigan DL, Witt SA, Glass B, Chung SE, Ellen J. (2006). Perceived neighborhood social cohesion and condom use among adolescents vulnerable to HIV/STI. *AIDS and Behavior*; 10 (6): 723-9.
- Sieverding JA, Adler N, Witt S, Ellen JM. (2005). The influence of parental monitoring on adolescent sexual initiation. *Archives of Pediatrics and Adolescent Medicine*; 159 (8):724-729.

Resource Guides:

- Lezin, N, Witt, S, Taylor J, & Bliesner, M. (2010). *Providing Access to HIV Testing Through Schools: A Resource Guide for Schools*. Scotts Valley: ETR Associates.
- Witt S, Sanchez-Cesareo M, Francisco VT, Willard T, Ziff M, Doll M, Chutuape K, Cooper-Walker B, Robles-Schrader G, Ellen J. (2005). Strategic Planning Guide for Connect to Protect ®.
- Witt S, Ellen J. (2004). Connect to Protect ® Structural Changes to Prevent HIV: A Compilation of Potential Strategies. Johns Hopkins University.

*Maiden name Witt

PRESENTATIONS, TRAININGS and WORKSHOPS

Keynotes:

- Guinosso, S (April 2015). *Survive or Thrive: Using Lessons from Neuroscience to Re-envision our Work with Adolescents*. Keynote presented at the American Teens in Crisis Conference, Valparaiso, IN (150 participants).
- Guinosso, S. and Christopher, D. (September 2014). *The Science of Learning*. Keynote presented at the PREP Regional Training, Salt Lake City, UT. (100 participants).
- Guinosso, S. (May 2014). *Positive Youth Development and Sexual and Reproductive Health*. Keynote presented at the PREP Regional Training, Washington, DC. (100 participants)

Guinosso S. (March 2013). *Adolescent Brain Development and Implications for Public Health*. Keynote presented at the PREP Regional Training, Los Angeles, CA. (75 participants)

Guinosso S. (August 2012). *Adolescent Brain Development and Implications for Public Health*. Keynote presented at the PREP Regional Training, Washington, DC. (75 participants)

Guinosso S. (July 2012). *Adolescent Brain Development and Implications for Public Health*. Keynote presented at the PREP Regional Training, Denver, CO. (75 participants)

Workshops:

Guinosso, S. and Christopher, D. (September 2014). *Masterful Facilitation*. Workshop presented at the PREP Regional Training, Salt Lake City, UT. (100 participants).

Guinosso, S. (October 2014). *Bounce Back: Fostering Youth Resiliency*. Workshop presented at the Health Teen Network Conference, Austin, TX. (60 participants)

Guinosso, S. and Christopher, D. (June 2014). *The Social-Emotional Adolescent Brain*. Workshop presented at the Teen Pregnancy Prevention Initiative Conference through the Office of Adolescent Health, Centers for Disease Control and Prevention and Family and Youth Services Bureau. (100 participants)

Guinosso, S. and Peterson, A. (June 2014). *Bounce Back: Fostering Youth Resiliency*. Workshop presented at the Teen Pregnancy Prevention Initiative Conference through the Office of Adolescent Health, Centers for Disease Control and Prevention and Family and Youth Services Bureau. (100 participants)

Guinosso, S. (May 2014). *Bounce Back: Fostering Resiliency Among Youth through Adolescent Pregnancy Prevention Programs*. Workshop presented at the PREP Regional Training, Washington, DC. (60 participants)

Guinosso S. (August 2013). *Adolescent Brain Development and Implications for Public Health*. Workshop presented at the California Maternal, Child and Adolescent Health State Conference, Sacramento, CA. (60 participants)

Guinosso S. (May 2013). *Tips and Tricks of the Training Trade – Effective Training and Facilitation*. Workshop presented at the Family and Youth Services Bureau's Annual Conference, Baltimore, MD.

Guinosso S., Christopher D. (May 2013). *Adolescent Brain Development and Implications for Public Health*. Workshop presented at the Office of Adolescent Health Annual Conference, Washington, DC. (40 participants)

Guinosso S. (February 2013). *Learn, Inspire, Persuade: the Art of Communicating and Reporting Evaluation Results*. Workshop presented at the PREP Regional Training, Atlanta, GA. (60 participants)

Guinosso S. (June 2010). *Survive Outside: HIV Prevention in Juvenile Justice Settings*. Workshop presented at the Kansas Department of Education State Conference, Kansas City, KA. (40 participants)

Sanchez-Cesareo M, Guinosso S. (October 2005). *Introduction to Logic Models*. Workshop at the Adolescent Medicine Trials Network Meeting, Rockville, MD.

Trainings:

Guinosso S. (July 2013). *Learn, Inspire, Persuade: the Art of Communicating and Reporting Evaluation Results*. Training presented at the Colorado Department of Education, Colorado Springs, CO. (30 participants)

- Guinosso S, Sanchez-Cesareo M. (April 2006). *Strategic Planning for Structural Change.* Training for the Connect to Protect coalition at the University of California, San Diego. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (March 2006). *Strategic Planning for Structural Change.* Training for the Connect to Protect coalition at Children's Hospital Boston, MA. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (January 2006). *Strategic Planning for Structural Change.* Training for the Connect to Protect coalition at Children's Hospital Los Angeles, CA. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (December 2005). *Strategic Planning for Structural Change.* Training for the Connect to Protect coalition at the University of Maryland. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (December 2005). *Strategic Planning for Structural Change.* Training for the Connect to Protect coalition at the University of California, San Francisco. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (November 2005). *Strategic Planning for Structural Change.* Training for the Connect to Protect coalition at DePaul University, Chicago, IL. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (August 2005). *Buying Upstream: Stopping the AIDS Epidemic at the Source.* Training for the Connect to Protect coalition at the University of Miami, FL. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (July 2005). *Buying Upstream: Stopping the AIDS Epidemic at the Source.* Training for the Connect to Protect coalition at DePaul University, Chicago, IL. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (June 2005). *Buying Upstream: Stopping the AIDS Epidemic at the Source.* Training for the Connect to Protect coalition at the Children's Hospital of Philadelphia, PA. (30 participants)
- Sanchez-Cesareo M, Guinosso S. (May 2005). *Buying Upstream: Stopping the AIDS Epidemic at the Source.* Training for the Connect to Protect coalition at the University of Puerto Rico. (30 participants)
- Guinosso S, Sanchez-Cesareo M. (April 2005). *Buying Upstream: Stopping the AIDS Epidemic at the Source.* Training for the Connect to Protect coalition at the University of Maryland. (30 participants)

Webinars and e-Learning Events:

- Guinosso, S. (April 2014). *Adolescent Brain Development and Implications for Public Health.* Webinar presented to California Office of Family Planning Grantees. (100 participants).
- Guinosso, S. and Coyle, K. (March 2014). *Disseminating Innovative Strategies.* Webinar presented to FYSB PREP grantees. (100 participants); available at: <http://www.acf.hhs.gov/programs/fysb/resource/disseminating-innovative-strategies-20140306>
- Guinosso, S., Wright, T. and Christopher, D. (February 2014). *Effective Delivery of Technical Assistance.* Online Training presented to North Carolina Department of Education. (20 participants)

Guinosso S. (September 2013). *Planning for Sustainability*. Webinar presented to FYSB PREP grantees. (200 participants); available at:
<http://www.acf.hhs.gov/programs/fysb/resource/webinar-20130918>

Poster Sessions:

Guinosso, S. (July 2014). *Adverse Childhood Experiences and Cognitive Development Among School-Age Children in the U.S.* Jack Kent Cooke Scholar's Weekend, Lansdown, VA. (400 attendees)

SERVICE

Lemonade: A Yoga Program for Youth **2013-present**

- Yoga teacher, teach yoga to adolescent males in the San Francisco Juvenile Hall

San Francisco Unified School District **2012-2013**

- Yoga teacher, taught yoga to middle school girls at Horace Mann Elementary School

Johns Hopkins Neuroscience and Public Health Consortium **2011-2012**

- Student representative, contributed towards development of a multi-institution online curriculum bridging neuroscience and public health

Johns Hopkins Faculty Search Committee **2011**

- Student representative, assisted with search for the new Chair of the Center for Adolescent Health

Johns Hopkins Departmental Student Association **2011**

- President, organized student activities and served as liaison between students and the department

Juvenile Justice Trainers Association **2010-2012**

- Board member, contributed towards conference planning for juvenile justice professionals nationwide

Health Initiatives for Youth **2006-2009**

- Board member, chaired 2007 and 2008 fundraising campaigns